SURGERY, GYNECOLOGY AND OBSTETRICS

AN INTERNATIONAL MAGAZINE, PUBLISHED MONTHLY

VOLUME XVIII FEBRUARY, 1914 NUMBER 2

A PRELIMINARY REPORT ON A SIMPLE AND RAPID METHOD OF PYLORIC CLOSURE IN GASTRO-ENTEROSTOMY [1]

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THE object of this communication is to report and place on record the results of a series of experiments undertaken to determine a rapid and safe procedure by which the pylorus can be temporarily or permanently closed during the operation of gastrojejunostomy. Ever since the employment of gastrojejunostomy in the treatment of gastric or duodenal ulcer, surgeons have recognized the desirability, under certain conditions, of effecting a closure of the pylorus.

While considerable difference of opinion has prevailed and still prevails regarding the advantages of pyloric closure in the various conditions in which gastrojejunostomy is indicated, and while it is neither desirable nor necessary on this occasion to enter into a discussion of these problems, all surgeons of experience will agree that in at least one group of cases, namely in bleeding duodenal ulcers, closure of the pylorus is positively indicated.

In the past many methods have been suggested and practiced to bring about this result. The simplest of these is closure by means of a tightly-drawn constricting ligature, with or without preliminary contusion of the parts, as suggested by Kelling; but unfortunately this has been found to be insufficient, for both animal experiments and clinical observation in the human subject have proven that the closure thus produced is a temporary one, as the ligature quickly causes a necrosis of the constricted area, the proximal and distal peritoneal surfaces brought in contact by the application of the ligature quickly unite; and when the necrotic area separates the lumen is re-established. Closure of the pylorus by means of infolding sutures, as suggested by Moynihan and Kelling; by means of a submucous plastic operation, as advocated by Girard; by means of a pedunculated seromuscular flap from the anterior gastric wall; or by a ligature of free transplanted autoplastic material, as proposed by Wilms, have all been employed, in some instances with encouraging results. F. H. Tappeiner,[2] however, reports a series of animal experiments with all of these methods. All of his dogs lived with the exception of one, which died at the end of two weeks from pulmonary embolism.

The specimens from the successfully operated dogs were removed after four, seven, and ten weeks.

As a result of a careful study of these specimens, the author states that not even a temporary constriction of the dog's pylorus could be accomplished either with infolding sutures, according to Kelling's method, with the submucous plastic, according to Girard, or with a pedunculated plastic from the anterior gastric wall. The same remark ap-

[2] Beitr. z. klin. Chir., 1912.

[1] Presidential address, Clinical Congress of Surgeons of North America, Chicago, November 10, 1913.

Fig. 1. Stomach from dog described in Experiment 1 showing aluminum band completely covered by new-formed tissue.

plied to thread ligature after contusion of the pylorus, for the ligature is promptly expelled into the intestinal lumen, and this is rapidly followed by a re-establishment of the pyloric canal. Better results are obtained by thread constriction without contusion of the pylorus, for in these cases a longer time is required for the migration of the thread through the pyloric wall. At the instant, however, that its transit is completed, the pylorus again becomes distended, for no solid resistant scar is formed during the experiment.

Still, the author states, it should be kept in mind that the muscle of the gastro-intestinal wall is not so strong in men as in dogs, the tissues of which, according to Schloffer,

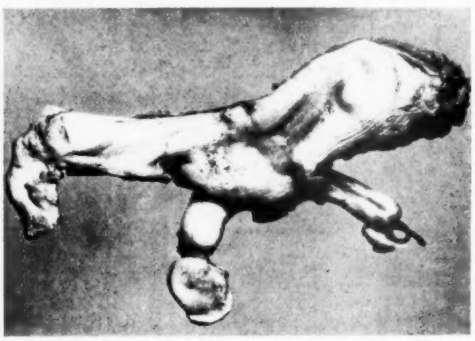

Fig. 2. Stomach from Experiment 2. Aluminum band in place, completely covered.

Fig. 3. Body and pylorus of stomach from Experiment 3 filled with water and tacked to a board, pylorus downward, showing no leakage.

possess a greatly superior capacity of regeneration; and it is possible that the threads take a much longer time to cut through in man as compared with dogs. Still better results are accomplished with the free transplanted strip of fascia, for this does not pass through the stomach wall but forms organic adhesions with it. Moreover, ligature with the strip of fascia can be completed almost as rapidly as with an ordinary thread, certainly much more rapidly than the gastric wall plastics.

As early as 1895, von Eiselsberg devised and practiced an operation which he designated *unilateral pyloric exclusion*. This consisted in a complete transverse division of the stomach proximal to the pylorus, and closure of the proximal and distal openings by Lembert sutures. This operation was originally devised to prevent irritation of inoperable carcinomatous ulcers of the pylorus by totally excluding from them the gastric secretions. Later he and his followers employed it in benign ulcers of the pyloric region as well as in ulcers of the duodenum.

That this method is universally successful is evidenced not only by von Eiselsberg's statistics, but by those of Haberer and others.

Fig. 4. Pyloric end of stomach, filled with water, showing no leakage from duodenum.

In the closing paragraphs of Tappeiner's report, the author remarks that he "believes himself justified in stating that the very best method of pyloric closure is undoubtedly the method of von Eiselsberg," although he recognizes certain disadvantages in this often prolonged procedure.

While all surgeons will agree that the method of von Eiselsberg can be relied upon to effect permanent closure in every instance, it will also be recognized that the procedure is necessarily a long one; and especially in cases of bleeding duodenal or gastric ulcers the patients are in no condition to withstand the added shock of such an operation. Moreover, there must also be considered the risk of infection where the gastro-intestinal tract is thus opened a second time.

Stimulated by the successful results of Halsted in effecting closure of the larger arteries by means of aluminum bands, the writer undertook a series of experiments in the Surgical Research Laboratory of the College of Physicians and Surgeons, to determine if this method could not be employed in pyloric closure.

Advantage was taken of the classwork in operative surgery, and days were chosen when the operation of gastrojejunostomy was demonstrated.

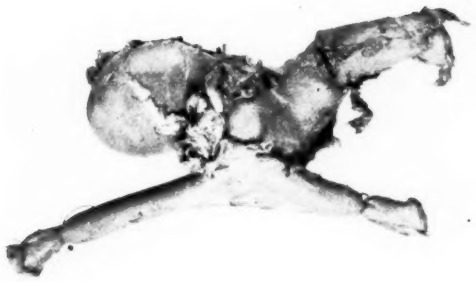

Fig. 5. Stomach from Experiment 4, after removal of band.

As soon as the anastomosis was completed, the pylorus was exposed and a closed Kocher's forcep passed beneath the pylorus through the mesentery. The forceps were next opened to enlarge the mesenteric rent, and a strip of sheet aluminum 1 cm. in width and about 5 cm. in length was drawn backward with the forceps through the mesenteric opening. This was next rolled with the fingers snugly around the pylorus, with sufficient compression to obliterate its lumen. The abdomen was then closed by layer suture. The application of the band in this manner took less than a minute, and while it effectively closed the pylorus, did not compromise the vitality of the tissues, as the following record of the experiments will show.

Experiment 1. Laboratory No. 75. After a posterior gastrojejunostomy by the suture method,

Fig. 6. Transverse section of restored pylorus, Experiment 5.

Fig. 7. Stomach from Experiment 6 showing aluminum band only partly hidden by new-formed tissue. Band applied 2 cm. proximal to pylorus. Dog killed on ninth day after operation.

an aluminum band was passed around the pylorus by the method just described and tightly molded into a circular constricting ring. Abdomen closed by layer suture, a dressing applied and the dog placed in the hospital. No marked reaction followed the operation, and the dog was discharged to the kennels in three weeks. Two months and ten days from the operation the animal was killed. Autopsy showed the band to be completely covered by newly formed tissue with but few adhesions. Stomach and a portion of the duodenum, with the ascending and descending portions of the jejunum, removed and

Fig. 9. Stomach from Experiment 8. Band incompletely covered after seven days.

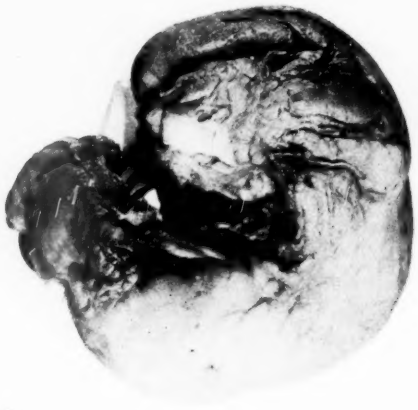

Fig. 8. Stomach from Experiment 7. Band covered.

freed from omentum. Fig. 1 shows this specimen as it was removed at autopsy.

Experiment 2. Laboratory No. 76. After gastrojejunostomy, band applied to pylorus in same manner as in experiment. No untoward symptoms during convalescence. Dog discharged to kennels in five weeks. Two months and ten days later dog killed. Autopsy showed band completely buried and invisible. Few adhesions. Fig. 2 represents this stomach after removal and after the attached omental tissue was dissected away.

Both of these specimens were preserved intact for laboratory demonstrations.

Experiment 3. Laboratory No. 77. Same procedure as in other two experiments. Dog died on twelfth day of pneumonia. Autopsy showed no evidence of peritoneal infection. Band completely covered and invisible. Small dark area just distal to band on anterior surface of duodenum, thought to

Fig. 10. Stomach from Experiment 9. Band about antrum removed at second operation.

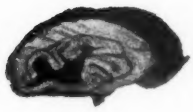

Fig. 11.

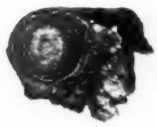

Fig. 12.

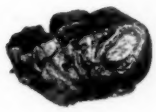

Fig. 13.

Fig. 16.

Fig. 11. Section through pyloric antrum after removal of aluminum band at second operation, showing lumen restored, Experiment 9.

Fig. 12. Transverse section of occluded pylorus; aluminum band in place, Experiment 3.

Fig. 13. Transverse section of occluded pylorus, aluminum band in place, Experiment 4.

Fig. 16. Gross appearance of pylorus closed by aluminum band, from proximal or stomach side. Experiment 3.

be necrotic, but on microscopic section and examination found to be simply a small subserous ecchymosis. After removal of the omental tissue and ligation of the ascending and descending loops of the attached jejunum, the fundus was cut across and the viscus filled with water to see if any leakage through the closed pylorus could be detected. The closure was found to be complete.

Experiment 4. Laboratory No. 78. Same procedure as in other animals. Dog died on nineteenth day from gastric dilatation. Autopsy showed band completely covered and invisible. Distal loop of jejunum obstructed by valve-like fold of mucous membrane. Stomach dilated and partly filled with cloudy fluid. After removal of the stomach from the abdomen, it was cut across about 12 cm. proximal to the pylorus. The water test as described in the last experiment was applied. No fluid escaped from the duodenal opening. Figs. 3 and 4 represent these specimens filled with water, tacked to a board, duodenal extremity downward, showing no leakage.

Experiment 5. Laboratory No. 300. Pyloric band applied as in other animals. No reaction. Dog remained well sixty-seven days, when second operation was performed. Abdomen reopened, band found completely hidden by newly formed tissue. This was incised and the band easily removed. Abdomen closed by layer suture. No reaction following the second operation, the dog was discharged to the kennels in eleven days. Several days later dog killed. Autopsy showed pylorus apparently patent. Stomach removed and freed of omentum.

Fig. 5 represents posterior view of stomach after removal. Pylorus cut through showing restoration of lumen with no evidence of necrosis of mucous membrane or muscular coat at site of previous constriction. Fig. 6 represents cut section through the now open pylorus.

Experiment 6. Laboratory No. 391. Same procedure with the exception that band was applied to pyloric antrum about 2 cm. proximal to pylorus. Somewhat heavier material used, and more force applied to effect constriction. Dog suffered from

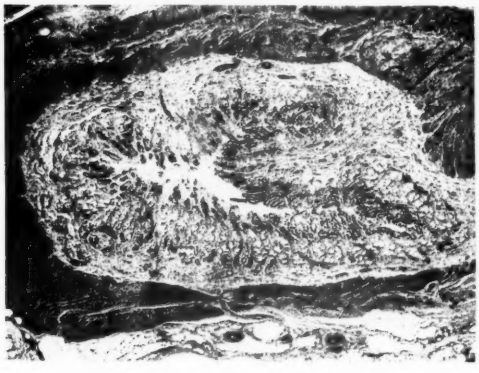

Fig. 14. Microscopic section of closed pylorus in Experiment 3 (low power).

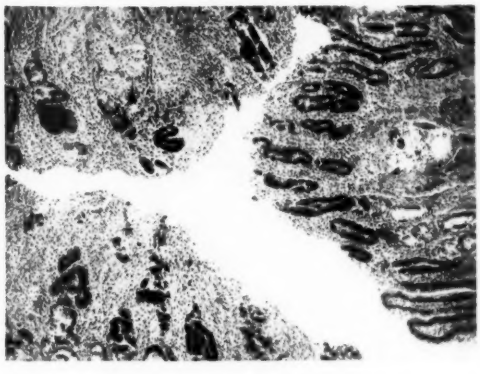

Fig. 15. Microscopic section (higher power) Experiment 3.

distemper and was killed on ninth day. No peritonitis. Band partly covered. Superficial wound of stomach by edge of band in repair. Complete stenosis demonstrated by water test (Fig. 7).

Experiment 7. Laboratory No. 392. Pyloric band applied in usual manner. Dog became ill and died with signs of sepsis. Autopsy showed pneumonia and peritoneal exudate, but no sign of leakage. Band completely covered. Dark area near band due to subperitoneal ecchymosis (Fig. 8).

Experiment 8. Laboratory No. 393. Stomach antrum occluded by band as in Experiment 6 2½ cm. from pylorus. Died in seven days of pneumonia. On autopsy, band was found only partly covered by new tissue. No signs of necrosis (Fig. 9).

Experiment 9. Laboratory No. 435. Anterior gastro-enterostomy. Band applied about 2 cm. proximal to pylorus. No untoward symptoms followed, and nineteen days later the abdomen was reopened and the band, which was completely hidden, was removed as in Experiment 5. No reaction followed the second operation. Dog subsequently killed. Autopsy showed pylorus apparently open and free from signs of pressure necrosis.

Fig. 10 shows anterior view of this stomach after removal. Fig. 11 shows cut section through previously constricted area.

The stomachs in Experiments 3 and 5 which had been proven to be occluded by the water test were further examined by sawing through the band and occluded areas. Figs. 12 and 13 show the appearance of the cut surfaces and Figs. 14 and 15 the microscopic appearances from sections cut after removal of the aluminum band from the tissues.

These sections show no evidence of necrosis of the mucous, submucous, muscular, or serous tissues. Fig. 16 shows the gross appearance of the occluded pylorus viewed from the proximal or stomach side.

A review of these experiments will show that the aluminum band produced a mechanical closure of the pylorus in every instance; that in every experiment in which the animal lived more than nine days the band was completely embedded in newly formed tissue and invisible; and that in none of the animals did the application of the band give rise to necrosis of the compressed tissues, signs of irritation or leakage.

NEOPLASMS OF THE RENAL PELVIS WITH ESPECIAL REFERENCE TO TRANSPLANTATION IN THE URETER AND BLADDER[1]

By WILLIAM E. LOWER, M. D., Cleveland, Ohio

PRIMARY tumors of the renal pelvis are very rare. Watson and Cunningham (1) report that Israel found only 2 cases of kidney pelvis tumors as compared with 68 in the kidney parenchyma, and that there was but one tumor (a papilloma) in 94 cases of renal and perirenal growths recorded at the Massachusetts General and Boston City Hospitals in 10 years. In 1904, Albarran and Imbert in their book, "Traité des Tumeurs du Rein," found only six instances in all of the literature on the subject. Stüsser (2), in 1912, reported a considerable number of tumors of the renal pelvis and a small number in which the ureter and bladder were also involved.

To the number of recorded cases in which there was transplantation into the bladder I desire to add another which came under my observation within the last year.

An unmarried woman, 63 years of age, was referred to me by her family physician, Dr. I. J. Wright, on August 5, 1912, because of hæmaturia.

Family history. Father died at the age of 84, mother at age of 83. The family history was negative for cancer, tuberculosis and goiter.

Personal history. She has had the usual diseases of childhood. Scarlet fever at age of 6. Never had typhoid fever or pneumonia. General health has been good until within the last three or four years. She has had no cough, generally a good appetite. Has had two gastro-intestinal upsets. Menses began at age of 11, have always been regular and not painful. Menopause at age of 53. Has had no vaginal hæmorrhage since. Five years ago patient strained her back in lifting. Following this she had considerable pain and soreness in back, which gradually wore off.

Present illness. For the past three years patient has passed bloody urine at various times. The first time she noticed the bleeding was after an acute retention caused by a large blood clot which obstructed the urethra. She has lost considerable blood in the past three years. Has had some dull pain in the back; this was perhaps more noticeable in the region of the left kidney. At the time of examination she was quite pale and weak.

Physical examination showed a well-nourished person with rather white skin, mucous membrane pale, but free from scars, eruptions or pigmentations. Vision was poor in the left eye, pupils were equal and regular and reacted to light and color. Some slight enlargement of the submaxillary and submental glands. No tumors palpable in the breasts. Lungs were resonant throughout and showed good excursions at the bases. Precordial area did not bulge and no thrills or friction rubs palpable. Heart about normal in size and the sounds were distinct; no murmurs discernible. Pulse was regular, rhythmic and of good volume; 90 per minute. No apparent enlargement of the liver. Both kidneys were palpable and the left somewhat tender. Extremities were symmetrical and reflexes active.

Cystoscopic examination. Bladder capacity 150 ccm. Introduction of the cystoscope easy. Situated just to the left of the left ureteral orifice was a dark tumor mass about the size of a hickory-nut. Upon closer inspection this was apparently an organized blood clot. It was adherent to the mucous membrane and could not be detached by the beak of the cystoscope.

After observing the rest of the bladder, which was found free from growth, stone or ulcer, and then reinspecting the tumor, a small amount of blood was seen upon the mucous membrane in that locality. My first impression was that the bleeding came from the base of this clot, but after a little longer inspection blood was seen to be spurting at irregular intervals from the left ureteral orifice. The urine from the right ureter was clear. The diagnosis of left renal hæmaturia was established but the exact cause undetermined. An exploratory operation was advised but declined at this time. As there was a possibility of this being a so-called essential hæmaturia injections of serum were recommended. The patient returned to her home and I heard nothing more from the case until October 19th, when she returned stating that she had been bleeding more or less continuously since her previous visit. She had had several injections of the serum without any definite benefit. Had lost considerable in weight and seemed paler and much more feeble than at the previous examination.

She was sent to the Lakeside Hospital and on October 21st another cystoscopic examination was made.

At this time the bladder capacity was 300 ccm. Introduction of the cystoscope easy. The dark tumor which had been observed at the previous examination was still present and it was plainly to be seen that this blood clot was adherent to a growth, only a small part of which was visible beneath the clot. In addition to this tumor, which had been observed at the previous examination, there was another growth about one-half the size of the larger tumor, situated upon the other side of the

[1] Read before the American Association of Genito-Urinary Surgeons, Washington, May, 1913.

Fig. 1. Growth in bladder, removed at time of first operation.

left ureteral orifice about one-quarter of an inch from the opening, as shown in Fig. 1. This was papillomatous in appearance, and was not bleeding. The ureters were catheterized, urine flowed freely from the right side, but only a small amount of turbid urine was obtained from the left side. Phenolsulphonephthalein intravenously appeared on the right side in three minutes and at the end of fifteen minutes 25 per cent was recovered. At this time there was no discernible bleeding from the ureter nor from either tumor. X-ray pictures of the kidneys and ureters were negative.

From the history of the case and from the previous examinations there seemed to be evidence enough to warrant an exploratory operation on the left kidney before undertaking to remove the tumors from the bladder, although there remained some doubt as to whether or not I might have been mistaken in the source of the bleeding. The tumors in the bladder were sufficient cause for the hæmaturia and yet the length of time that the patient had observed blood in the urine, the slight tenderness in the region of the left kidney and the appearance of the second tumor since the first examination, led us to speculate on the possibility of a tumor of the renal pelvis with transplantation into the bladder.

Operation. As she had stopped bleeding, the operation was delayed for a number of days, hoping that she might gain some in strength and be a better operative risk. Her hæmoglobin at this time was 60. On October 26th, under gas anæsthesia, with local infiltration of novocaine, the usual oblique incision was made for the exposure of the kidney. The kidney was found to be somewhat enlarged and the pelvis very much distended (Fig. 2). Palpation did not reveal any concretions or solid tumor, but a doughy mass could be detected. We did not consider it advisable to open the kidney pelvis lest we might infect the wound with the contents. The kidney was removed and the ureter ligated at a point about 3 inches from the kidney. At the point of division of the ureter there was no evidence of growth. The wound was closed with chromic catgut and the small cigarette drain inserted.

The patient was then placed upon her back and slightly in the Trendelenburg position. A transverse incision was made above the pubis, the recti muscles separated, the bladder exposed and opened. The tumor containing the organized blood clot, at the base of which was a papillomatous growth, was removed; it was about the size of a filbert. The point of excision was sutured with oo chromic catgut.

The other tumor was much smaller and much softer. It was removed in a similar manner and the wound closed. No other evidence of tumor was found anywhere in the bladder. The opening into the bladder was completely closed without drainage. The patient stood the operation well and there was no change in the pulse rate.

Post-operative history. On October 28th the wound was inspected and the drain removed from the kidney wound. It was clear and apparently healed by first intention. Patient was comfortable and voiding easily. The urine was bloody. On the 30th the clips holding the skin were removed. Patient was still passing bloody urine. This was difficult to account for but thought to be due to bleeding from the base of the tumors which had been removed from the bladder. On October 31st urine was still bloody. Bladder washed with boracic acid and adrenalin solution injected. November 2d hæmorrhage from the bladder very slight, general condition of the patient good. She was discharged November 23d in very good condition and feeling very comfortable. On January 21, 1913, patient telephoned me from her home in a near-by city that she was again having hæmorrhage from the bladder. She reported at the office on the 22d. She had gained considerably in weight, her color was much improved and she stated she felt better than she had for a number of years.

Cystoscopic examination No. 3. Bladder capacity 150 ccm. Introduction of the cystoscope easy. Extending from the right ureter and folding back on each side was a growth (Fig. 3). It was evident that this growth came from within the ureter and seemed to be pushing down and out of the ureteral opening as shown in the illustration. It was quite apparent that the ureter was involved and a second operation advised for its removal. She returned to the hospital and on January 28, 1913, was again operated upon under nitrous oxide anæsthesia and novocaine infiltration.

Incision was made extending from the lower end of the old scar through which the nephrectomy had been done, uniting with the scar for removal of the bladder tumors. The ureter was dissected from the scar above and completely isolated to the entrance into the bladder and a part of the bladder excised, as shown in Fig. 4. Bladder was closed and a small drain inserted down to the point of excision of the

Fig. 2. Photograph of kidney showing distended pelvis.

growth in the bladder. In removing the part of the bladder around the ureteral orifice the peritoneum was opened accidentally and immediately closed with plain catgut.

Cystoscopic examination No. 4, February 14, 1913. Bladder capacity 300 ccm. Around the region of the former ureteral orifice was a small mass of scar tissue and some inflammatory tissue. At the bottom of the bladder were a few small hæmorrhagic spots, otherwise the bladder mucosa seemed perfectly normal. Upon vaginal examination a small mass could be palpated in the region from which the left ureter had been removed. The mass was slightly tender and seemed to be inflammatory rather than a recurrence.

Cystoscopic examination No. 5, March 6, 1913. Bladder capacity 300 ccm. In the region of the former ureteral orifice there was only a contracted scar, the œdema which had been present at the previous examination had entirely disappeared, but there still remained a number of these small hæmorrhagic spots. The exact nature of these could not be definitely determined, but there was a strong suspicion that they might be the beginning of a recurrence.

Cystoscopic examination No. 6, April 1, 1913. At this time the bladder held 150 ccm. and situated just behind the trigonum near the area of excision of the left ureteral orifice was a small tumor mass. This was cauterized with the high frequency cautery on April 2d, and again on April 8th and 19th. At this time the bladder seemed to be in a nearly normal condition, with almost entire disappearance of the tumor, there remaining only a white slough at the site of the growth. The pathologic report of the growth in the kidney pelvis, ureter and bladder was identical, all three being malignant papilloma, as shown in Figs. 5, a, b, and c.

At this writing there is no evidence of recurrence of the growth in the region of the kidney or ureter. I am inclined to believe that the tumor at no time had gotten outside of the mucosa.

Upon opening the kidney pelvis it was found to contain considerable amount of broken down material with fringed edges along the calices, as shown in shaded part in the diagrammatic illustration (Fig. 6). The ureter contained a number of pâtches of the growth, but greater in abundance near the lower end of the ureter where they seemed to pile up at the constriction of the ureter into the bladder.

Microscopic section of the ureter at the point of excision of the kidney was free from any evidence of growth.

No definite theory is advanced for the cause of these neoplasms. Albarran and Imbert (3) found that in 15 per cent of their cases calculi were present and concluded that they had produced chronic irritation and thus furnished a good field for the neoplasms.

Fig. 3. Growth in bladder removed at second operation.

They usually occur between the ages of 40 and 60 years and are more frequently found in men.

As for the symptoms, they are quite variable and depend as a rule upon the stage of development. Hæmorrhage, tumor, pain, and elements in the urine generally indicate that a growth is present. The first sign is usually hæmorrhage. It may be continuous, intermittent and occasionally cases are noted in which it is absent entirely. It may also vary in amount, at times being scarcely noticeable and then again so profuse as to cause death. Cumston (4) noted that in cases of intermittent hæmotonephrosis one could usually suspect tumor of the renal pelvis. He also observed that when there is hæmaturia without retention, it points as a rule to a growth in the upper part of the renal pelvis or calices and large hæmatonephroses usually indicate neoplasms in the renal pelvis close to the ureteral orifice.

Pain is not a constant symptom nor an important one. It may come in attacks of colic, unexpectedly, and occasionally there is merely a sensation of numbness in either the opposite leg or in the loin and leg of the same side. Hartmann (5) reports a case of a man, 51 years old, who had suffered almost continuously with renal pains since his twentieth year. Another instance of long renal colic duration is Oraison's (6) case. It concerns a woman, 50 years old (at the time of report), who complained of violent renal colic from her twenty-second to her fortieth years. The

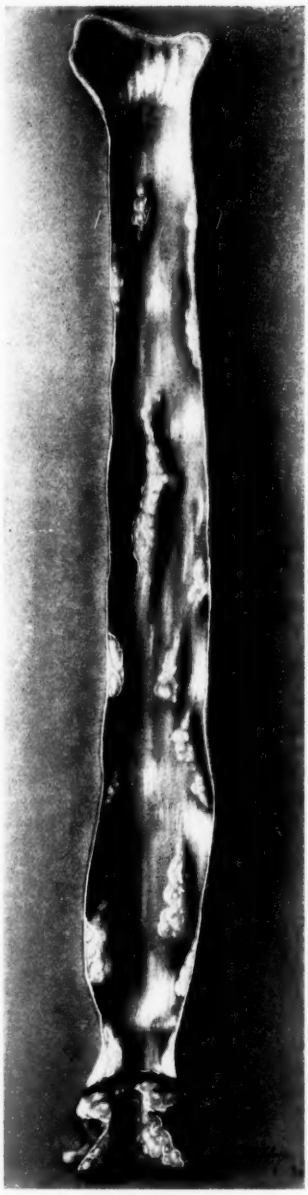

Fig. 4. Papillomatous growth in ureter and left ureteral orifice.

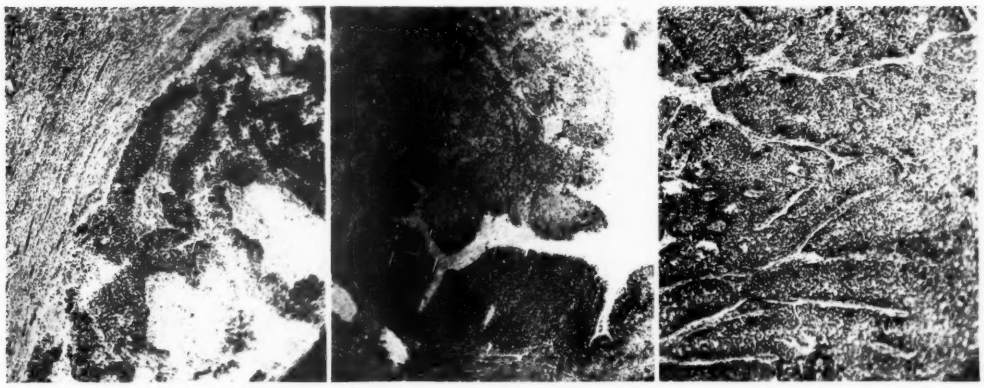

Fig. 5 A. Fig. 5 B. Fig. 5 B.

Fig. 5 A. Section of growth from the kidney pelvis.
Fig. 5 B. Section of growth from the ureter.
Fig. 5 B. Section of growth from the bladder.

pain occurred at intervals of one to three days and as a rule was followed by hæmaturia lasting several days. After she was 40 the pains were less severe but continuous.

There are also some general symptoms to be noted, such as weakness, marked pallor, loss of flesh, anæmia, and loss of appetite. Fenwick (7) has called attention to an important sign, namely, that when there is a growth in the pelvis of the kidney in which there is pelvic dilatation the ureteral orifice is altered in the nature of a dull-colored swelling and elongation of the opening of the ureteral orifice. This was not noted in my case.

It is very often hard to determine just where the growth is located, but with the aid of the cystoscope and by ureteral catheterization and injection of collargol one may make a fairly accurate diagnosis. A tumor at the ureteral orifice should lead one to suspect a growth higher up. It is not unlikely that in the recurrent tumors of the bladder, especially those situated in proximity with a ureteral orifice, the real source of the trouble is a growth in the ureter or kidney pelvis and the recurrences are transplants. Evidences of renal pelvis papilloma may be present for a long time, even months or years in a benign form, and the general health may suffer only slightly.

Sooner or later, however, it will show symptoms of malignancy. Early removal of the kidney, and I believe in most cases the ureter as well, is the only treatment.

Herewith are appended the cases of a similar condition. Case 17, reported by Picot, is of striking similarity.

Case 1. Busse's (8) case in a man, age 43, who had a sudden attack of hæmaturia and pain in the left renal region. In the following seven years they did not entirely disappear. In his fiftieth year bladder hæmorrhages brought him to the hospital. Nephrectomy was performed by Prof. Schuchardt. The patient recovered, but died of exhaustion four and one-half months after the operation. The inner surface of the renal pelvis was covered with fine villi, which reached to the ureter. There was a tumor in the bladder. The edge was covered with numerous long villi. At first these were benign, but a few months before death became malignant.

Case 2. Kohlhardt's (9) case of a woman, 60 years old. For two years she had noticed blood in the urine intermittently. The left kidney was extirpated and a villous tumor was found partly reaching into the ureter. The renal pelvis and calices were much enlarged. In the upper half of the former, close to the ureter end, was a tumor mass (the size of a small pigeon egg) which inclined toward the ureter. Small villous tumor masses were located on the entire mucous membrane of the renal pelvis and calices to the upper third of the ureter. Briefly stated, there was a multiple villous tumor formation originating in the left renal pelvis, renal calix and ureter, the bases of which showed proliferation of the same. As a result of the villi, the urine flow was hindered and hydronephrosis resulted. The patient was cured. Stüsser (10), in his account of the case, states that Kohlhardt excluded

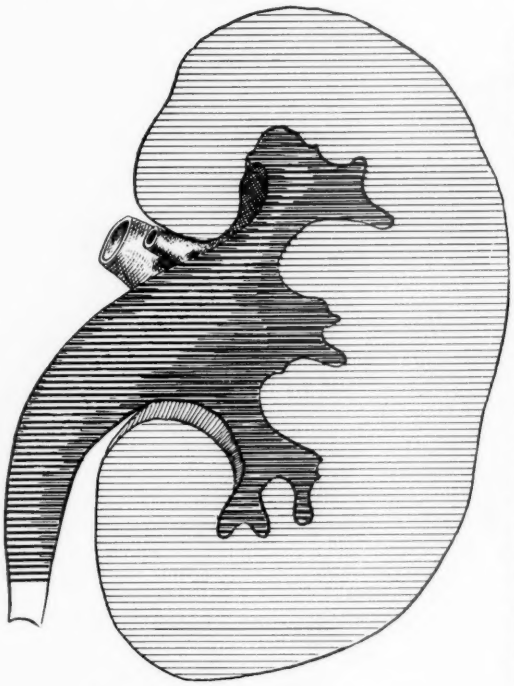

Fig. 6. Diagram showing growth in pelvis.

carcinoma and inclined toward the diagnosis "fibroma papillare diffusum" and "pyelitis villosa," and also that the patient died of regionary metastases one and one half years later.

CASE 3. Poll's (11) case of a man, 41 years old. Three and one-half cm. of the ureter was removed, also the left kidney. The renal pelvis was enlarged to the size of a man's fist. Small groups of gray-white protuberances were seen in the renal pelvis. Briefly stated, there was a villous tumor of the mucous membrane of the ureter and the renal pelvis of the left kidney, which did not penetrate deeply here, but did in the calices. He also had hæmorrhages. The growths extended toward the bladder. The specimens were examined after the death of the patient. Poll (12) says that years ago various names were used for these tumors, such as: villous polypi, villous cancers, tumores villosi, fibroma papillare, papilloma, without distinguishing between benign and malignant. To-day they go under the name of villous cancer or carcinoma papilloma. Due to reasons unknown to him, papillary growths existed on the mucous membrane of the renal pelvis and the ureter which showed plainly carcinomatous growths in the calices. It is not known positively whether they were benign or malignant at the start. It probably was carcinoma in the calix.

CASE 4. Rayer's (13) case of a woman, aged 58. She died of hæmorrhage. The autopsy showed a collection of fluid in the abdominal cavity. In the greatly enlarged liver were numberless cancer nodes of all sizes. The mesenteric glands were infiltrated with cancer. The kidney itself showed nothing. On the mucous membrane of the renal pelvis could be seen "small, pedicled tumors of gelatinous nature." Similar changes were also found in the ureter and bladder.

CASE 5. Drew's (14) case of a male, 56 years old, who was admitted to the hospital July 24, 1896, under the care of Mr. Barker. For three and one-half years at intervals he suffered pain, resembling renal colic. Other symptoms were hæmaturia, at times quite profuse, and a swelling in the abdomen, which was probably due to a large tumor, evidently renal in the left side of the abdomen. On July 30th nephrotomy was performed but no stone was found. The patient died November 11th. The wound discharged much pus, containing flakes of lymph which, under the microscope, were found to exist of renal epithelia and pus. The autopsy showed the right kidney slightly distended; the left much enlarged; the renal pelvis and calices greatly dilated; the lining membrane of the latter thickly covered with pinkish papillary growths; there were numerous spongy whitish nodules near the renal pelvis. The ureter was dilated and filled with pus. Along the length of the mucous membrane it was filled with tufts of delicate growths, attached by a narrow base. The bladder was small. A large red papillary growth was springing from the margin of the left ureteral orifice. In it was a cyst the size of a pea. The base of the villous growth did not infiltrate the wall of the bladder. Morris (15) in mentioning the case said that the growth in the renal pelvis was epithelial with cells of transitional type and those in the ureter were simple papilloma.

CASE 6. Mioni's (16) case. A man, 34 years old, had suffered with kidney colic four years. For two and one-half years he had painful hæmorrhage. The kidney upon palpation was very painful and the urine contained many erythrocytes, leucocytes and also numerous large cells of varied shape. The renal pelvis and calices were much enlarged. Both the kidney and ureter were removed to the bladder. The microscopic examination showed a typical papillomatous proliferation of the cylindrical epithelia of the renal pelvis and ureter. Several gland-like formations caused Mioni to diagnose the case as "adenocarcinoma papilliferum." Further progress of the case is unknown.

CASE 7. Matsuoka (17) reports a case of papilloma of the renal pelvis from the surgical clinic of Tokio. The diagnosis was "pyonephrosis" and nephrectomy was performed. A purulent cystic kidney with villous growths of the renal pelvis was found. The villi are transplanted to the ureter and microscopically are located upon a connective-tissue framework which is covered with a thick layer of epithelial cells. The description of this picture is

very interesting, since it shows the transition from the benign to the malignant stage.

CASE 8. Hedenius and Waldenstroem (18). The diagnosis showed bladder and kidney calculi. In the autopsy it was found that the renal pelvis was enlarged because the mucous membrane was covered with a mass the size of a hazel-nut; it was closely joined, uneven and composed of grayish-white, soft nodules. From the renal pelvis the neoplasms went to the ureter, the wall of which showed a thickness of 1.5 cm. In the histological pictures the neoplasms showed fine, connective-tissue stroma, rich in large epithelial cells of varied forms with large kernels and granular protoplasms. The bladder contained no stones.

CASE 9. Derewenko's (19) case of a woman, 54 years old, who had noticed blood in the urine since the summer of 1902. In March, 1905, she was taken to the clinic and operated on the 29th for hæmatonephrosis. The kidney was not removed. There was no trace of a new growth. The patient developed pneumonia, then a suppuration and was dismissed June 20th. In September of the same year she was received again. The diagnosis based on the previous operation was unilateral hæmorrhagic nephritis, and in October the left kidney was extirpated and the patient dismissed. In the hydronephrotic atrophied kidney were benign cauliflower new-growths. Blood was again noticed in the urine nine months later and the patient received into the hospital. At the cystoscopic examination a papillomatous formation was seen projecting from the ureteral orifice. It was radically extirpated by opening the bladder. Scarcely one-half year after her dismissal she complained of lumbago, paraplegia, retention of urine, paralysis of the bladder and rectum. Later gangrene set in and after a few days the patient died. The autopsy showed a tumor in the left renal region which destroyed the second and third lumbar vertebræ and caused metastases in the liver and lungs. This new growth was called villous cancer and it was emphasized that it was different from the new growths found before in the ureter and renal pelvis. Derewenko is of the opinion that the benign papilloma of the renal pelvis caused metastases in the retroperitoneal lymph glands at the hilum and in the aorta, which remained latent. The ureter was entirely covered with proliferations and only after removal of the kidney and ureter did these malignant proliferations begin.

CASE 10. Savory and Nash (20) report a case by Barker of a man, 56 years old who had villous carcinoma in the dilated calices and simple papillary tumors in the bladder and ureter.

CASE 11. Cattani's (21) case is that of a man 63 years old with the following history: Immovable tumor of the abdomen, hæmaturia with symptoms of cystitis and pyelitis; pains in the lumbar region; slight albuminuria; in the urine sediment were cancerous elements; cachexia, exitus. The cancerous infiltration originated at the beginning of the ureter; it was of grayish color and partly ulcerated. The tumor, which was transplanted to the renal pelvis and had advanced to the cortex of the kidney, was found to be a medullary carcinoma.

CASE 12. Bradford's (22) case of a male, age 44, who was brought to the hospital October 1, 1897, with a severe attack of delirium tremens and severe hæmaturia. Six months ago he noticed blood in the urine for one week. A large tumor was found in the left hypochondrium extending down to the umbilicus. The patient died October 5th. The post-mortem showed that the tumor felt during life was the left kidney. There was a calculus in the renal pelvis, the mucous membrane of which was thickly covered with papillary benign growths, which extended into the ureter one and one-half inches. There was no epithelial ingrowth into the wall of the ureter and renal pelvis. A stone caused hydronephrosis.

CASE 13. Busse's (23) case, a man 61 years old. Prof. Häckel extirpated the kidney tumor under great difficulties. The operation was apparently successful but the patient died four weeks later. He had an abscess in the lung, and also hæmorrhage. The wall of the renal pelvis was covered with small, wart-like elevations on the mucous membrane with broad base or thin thread-like pedicle; similar ones were also present all over the mucous membrane of the ureter, thickest near its orifice into the bladder, where a tumor the size of a hen's egg was found. There was a similar tumor in the bladder near the orifice of the right ureter and two smaller ones near the large tumor. That this was a benign tumor is known from the histological formation of the tumor, also from the patient's history. The suffering could be traced to twenty years.

CASE 14. Knowsley Thornton's (24) case of a woman 32 years old. She is said to have suffered with pain in the right renal region since she was eight years old. Nine years before admission to the hospital she had occasional attacks of hæmaturia. During the last few months a tumor in the right side of the abdomen had become very painful. She had a cystic kidney. Nephrectomy was performed and the patient was up on the 22d day. The specimen is unique; the ureter is surrounded at the exit from the kidney by a papillary growth the size of a pea. It is capped by an olive-shaped dark-brown calculus. The openings from the calices into the renal pelvis are dilated, two are blocked by these smaller brown calculi. The patient made good recovery. Stüsser (25) in his report of the case classes it among the benign. He said that upon examination of the specimen it was found that there was a villous new growth which extended from the mucous membrane of the renal pelvis at the beginning of the ureter and in other parts of the renal pelvis. There was no histological description of the papilloma and nothing more is known of the case.

CASE 15. Murchison's (26) case in a man aged 65 years. The patient had frequent hæmorrhage and pain. Other complications set in and the patient died. At the autopsy it was found that the

base and surrounding of both ureters, especially the right, were studded with long, delicate, villous processes, not forming a distinct tumor. The ureter and pelvis of both kidneys were enlarged. The mucous lining of the ureter was free. The case was diagnosed as villous disease of the bladder. In the kidneys the same morbid processes had begun. Stüsser (27) classes this case among the benign and adds that at the autopsy it was found that both renal pelves were covered with long, villous tumors and that in the lower part of both ureters kidney concretions were found.

CASE 16. Fenwick's (28) case of a man 70 years old, whose right kidney was removed on account of papillary growths in the renal pelvis and calices. He had suffered with hæmorrhage almost constantly for two years. At the end of the next year, a recurrence was noted in the right ureter and this was removed. Watson and Cunningham (29) in their report of the case state that the patient died uræmic four years later, and that toward the end there was carcinoma of the prostate and squamous-cell cancer, affecting the nephrectomy scar.

CASE 17. Picot (30) has recently reported a case of a man 53 years old who was brought to the hospital March 26, 1912. Seven years ago he had two hæmorrhages, the following year five or six attacks and lately they had become more frequent and lasted longer. He also complained of continuous pain on the left side. The diagnosis based on a cystoscopic examination was: three tumors of the renal pelvis with secondary transplantation in the bladder. Nephro-ureterectomy was performed. The ureter showed neoplastic infiltration and also two tumors having the same characteristics as those in the renal pelvis. All these papillary tumors macroscopically had the same aspect as the vesical tumors recognized by the cystoscope. The patient stood the operation well and returned home May 11th.

CASE 18. Israel's (31) case. Israel reports a case of a villous growth on the mucous membrane of the renal pelvis. Near the entrance to the bladder a new growth could be seen which covered the last 3 cm. of the ureter. The patient, a man 51 years old, had a severe hæmorrhage four years ago. Since then the attacks have been more frequent but less severe. He did not complain of pain nor colic, but by palpation it was felt that the kidney was enlarged. Retention of urine and the hæmorrhages had caused hæmatonephrosis. Nephro-ureterectomy was performed.

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THE FUNCTION OF THE PERIOSTEUM IN BONE TRANSPLANTS, BASED ON FOUR HUMAN TRANSPLANTATIONS WITHOUT PERIOSTEUM, AND SOME ANIMAL EXPERIMENTS[1]

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THOROUGHLY to appreciate some of the questions involved in the subject of bone transplantation, it will be necessary to consider briefly the three different views of recognized authorities as to the manner of regeneration of a graft.

1. The universally accepted view in Germany is that of Axhausen, who maintains that the bone in a graft always dies, is absorbed and is reformed from the periosteum, which alone remains living in transplants.

2. The second view is that of Macewen of Glasgow, who says that the bone in a graft is reproduced from the proliferation of osteoblasts, derived from the osteoblasts within the bone of the graft itself, and its regeneration takes place independently of the periosteum, whose only function is that of a limiting membrane, which prevents the spread of the osteoblasts into the surrounding tissues.

3. The third view is that of Murphy, who says that the graft is not osteogenetic but simply osteoconductive. Provided that it be in contact at one or both extremities with other living bone, the graft acts simply as a scaffolding for the growth of the capillaries with their osteogenetic cells as they advance from the living, contacting extremities into the graft. The periosteum takes no part in the actual reproduction of new bone. To analyze Dr. Murphy's transplantations a little further, I may say that I have gone through all of his reported transplantations with the object of finding out just what was done with the periosteum in each procedure. In a number of cases this was not stated. In all such instances I have ascertained from him in writing exactly what was done with the periosteum. In not a single instance did he omit to transplant grafts with more or less of their covering periosteum. From a practical point of view, I should judge that he does not know just what would have happened had he transplanted grafts without their periosteum, so that his conclusions as to the function of the periosteum are only conjectural.

The same may be said of Albee's work along this line. He agrees with Macewen and Murphy that the periosteum is unimportant so far as the life of the graft is concerned, yet, notwithstanding this view, he has never done a human transplantation without the periosteum, although he has performed 160 transplantations in all. It would strike the unprejudiced critic that the theories and practice of these men do not quite agree. Macewen and Albee have argued that, because they did not consistently obtain bone as a result of transplanting periosteum alone, consequently the periosteum has little function when it is left attached to grafts. Early in my work along this line it struck me that the proper angle from which to view this question of the function of periosteum could be obtained only from transplanting grafts of various sizes with and without their periosteum, since this is exactly what we do in our human transplantations. We do not transplant periosteum alone. The former is precisely what I have done and I think that I have definitely and conclusively proved, as you will later see, that the periosteum has a very important function in maintaining the nutrition of the graft.

When I looked over the results in a general way of both my animal and human transplantations, at first sight many of them seemed contradictory. On further analysis, however, it appeared as though all of these contradictions could be readily explained if we looked at it in this light: The life of a living graft, whether transplanted in the human

[1] Read in the Section on Surgery, American Medical Association, Minneapolis, June, 1913. From the Surgical Research Laboratory of Columbia University.

Fig. 1. Case 1. Rib grafted into a defect in lower jaw. A, rib.

being or in animals, whether transplanted with or without its periosteum, is probably entirely dependent on its being supplied with sufficient blood to keep it alive. I am inclined to believe that this will be the solution of the many vexed, theoretical considerations of bone grafting, and will explain the apparent contradictions and varying results obtained in bone transplantations. To particularize further, practically every bone graft made with its covering periosteum will live and grow if asepsis be attained. The question is, What will happen to grafts without their periosteum? The answer that I have obtained from my work along this line is this: The life of a graft without periosteum will depend entirely on its blood-supply. If it obtains sufficient blood, then it will live and grow; if not, then it will die. A small graft without periosteum will have a better chance of getting sufficient blood to its cells than a large one without its periosteum; but in either case there is always some doubt as to whether the graft will live, if it be without its periosteum. The periosteum on a graft does one of two things, or possibly both, namely:

1. By its presence it so favorably influences the nutrition of the graft, that is,

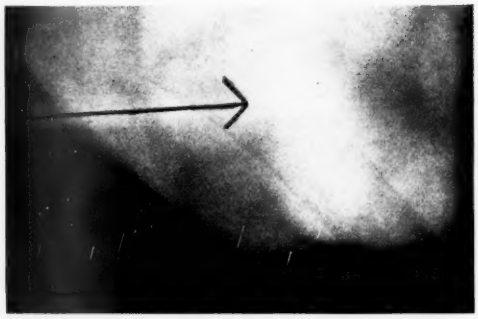

Fig. 2. Case 2. Rib grafted into inguinal hernia without periosteum.

increases the blood-supply to its cells, as to keep it alive, or

2. In case the bone cells in the graft die from insufficient nourishment, the periosteum supplies living cells to the graft, by means of which the bone is regenerated.

I can see no possible reason why the living cells in a graft should die if they be provided with sufficient blood. I hold this to be the function of the periosteum when attached to a graft. I hope to justify the soundness of my position in the illustrations.

HUMAN TRANSPLANTATIONS

CASE 1. In November, 1910, I removed half the lower jaw from a lad of 12 for a large giant-cell sarcoma. Fifteen months later I grafted into the defect in the lower jaw a piece of rib which was entirely stripped of its periosteum. One end of the rib was beveled and this was sutured to the freshened edge of the remaining half of the jaw. The wound healed by primary union. Fig. 1 shows the rib in position. Five months later, a röntgenogram showed an entire disappearance of the grafted rib. There never was any discharge from the scar.

The result in this case is a contradiction of Dr. Murphy's second conclusion, that "bone, with or without the periosteum, transplanted in the same individual and contacted at one or both ends with other living, osteogenetic bone, always becomes united to the living fragment, and acts as a scaffolding for the reproduction of new bone."

Had I split the rib longitudinally so as to afford a better blood-supply to the bone-cells of the graft, it seems to me that it might have lived, or had I transplanted the rib with periosteum covering half its surface.

Fig. 3 K, Case 3.

Fig. 3 P, Case 3.

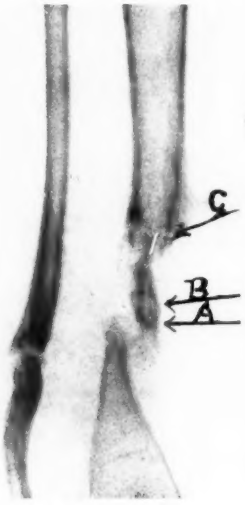

Fig. 4 B, Case 4.

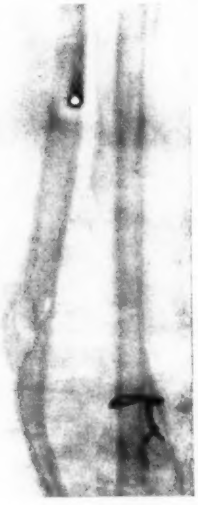

Fig. 4 E, Case 4.

A, section for microscopic examination taken from tibia; B, section for microscopic examination taken from graft; C, graft.

CASE 2. The patient was a man, aged 32, into whose inguinal canal I transplanted a piece of a rib to strengthen a hernial repair. Fig. 2 shows the rib in position. A röntgenogram four months later, showed that the rib has entirely disappeared.

This case is corroborative of Dr. Murphy's conclusion that bone transplanted into muscle always dies and is ultimately absorbed. That this statement is not always true, however, will be seen later from some animal experiments. Had I split this rib likewise, I believe that the consequent better blood-supply to its cells would have kept it alive.

CASE 3. The patient was a man, aged 32, who had received a compound fracture of the left humerus, a month before he came under my care. He was operated on immediately after the accident in another institution and the fracture was wired. This was followed by violent sepsis. Two sequestra were picked out of the wound the next day after the man came under my care. The patient had a musculospiral paralysis, indicated by the wrist-drop. The result of the application of a Lane plate to the fracture, after sawing off the ends of the fragments, was still non-union.

Five months after the previous plating, on account of non-union, I grafted into the medullary cavities of the fragments, which were first reamed out, a section from a subperiosteally resected rib. After the rib was in position, there was some side-to-side rocking of the fragments on each other, due to the fact that the rib was evidently a trifle too short. On this account I applied a Lane plate. It was then thought best to unite the divided musculospiral nerve. The ends were dissected out and it was found that there was a defect of an inch and a half to be bridged over. This was accomplished by cutting out of the distal extremity a segment, which was turned up to meet the proximal end, to whose freshened edge it was sutured with chromic gut. The result of this bone grafting was very disappointing. A picture (Fig. 3 K) after nine months showed no proliferation whatsoever of its bone and there was no better union than at the time of the grafting.

That the musculospiral nerve was regenerating was shown by the fact that sensation had returned in the distribution of the nerve, although there was no motor power as yet. Nine months after the rib grafting, I removed (Fig. 3 K) the remains of the grafted rib. A section of it under the microscope showed that it was undergoing rarefying osteitis and that it consisted mostly of dense fibrous tissue with scattered areas of bone. The medullary cavities of the fragments were reamed out (Fig. 3 P), and into them was inserted a graft, taken from the front of the tibia with periosteum on two of its sides. A nail was inserted in its upper end to prevent its riding up into the medullary cavity.

The result of this grafting was unfortunate. There was infection of the wound, requiring the removal of the nail and the dead graft. So that his condition was not improved.

CASE 4. This is an exceedingly interesting case of non-union in a birth fracture of both bones of the

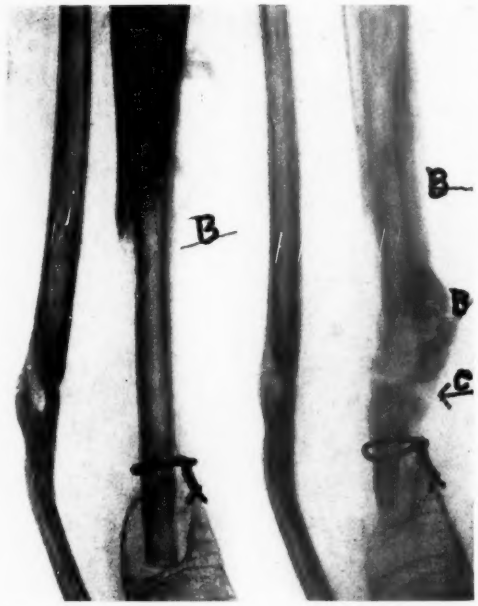

Fig. 4 I. Case 4. B, new bone in a thin plate along periosteum. Taken two months after operation, shows beginning new bone being formed.

Fig. 4 K. Case 4. B. new bone from periosteum; C, fracture.

leg in a child, aged 17 months. The non-union I attributed to insufficient immobilization of the leg. I cut down on the fracture and, after freshening the pointed extremities of the fragments, there resulted a defect of an inch and a half in the tibia, which made transplantation of bone necessary. A graft from the opposite tibia was chiseled out with its periosteum attached. The periosteum was then deliberately peeled off the graft with the object of confirming Macewen's view of its unimportance. One end of the graft was pointed and it was wedged into the medullary cavity of the upper fragment, and the lower side of the transplant was spliced to the side of the lower fragment with chromic gut. The subsequent union of the wound was by first intention. Fig. 4 B shows the condition six months after the grafting. It was very disappointing to see the tibial graft gradually melt away in the tissues, so that, after six months, it was not more than one quarter its original size. Since union was by first intention, there seems no way of accounting for the death of the graft save on the basis of a lack of periosteum, or of a deficient blood-supply, or both. Fig. 4 E shows the condition six months after the previous grafting operation, at which time I transplanted 5 inches of the opposite, healthy fibula into the medullary cavities of the fragments, as seen. It was transplanted with the periosteum covering its entire circumference, and this was not split in the slightest. The result shows that this splitting of the periosteum on the graft is entirely unnecessary and accomplishes nothing. Below, a wire-encircling suture was used to hold the graft in place. On the right one sees the defect made in the fibula. Fig. 4 I shows new bone being formed along the periosteum of the fibula graft, as a thin plate, two months after grafting.

Fig. 4 K was taken six months after the transplantation. To my mind it seems to imply the necessity of revising Dr. Murphy's conclusion that a graft is not osteogenetic in itself. A fracture has taken place in the mid-third of the transplant, due to the fact that the child kicked off the splint one night. We see that this fracture of the graft itself is consolidating nicely under an enormous callus. If we accept Dr. Murphy's conclusion, we must believe that all this callus came through the graft from the contacting old bone of the neighboring stumps.

I do not accept this view for a moment, but believe that the callus arose either from the periosteum or from the bone of the graft itself. Along the outer surface of the graft we see at least a quarter of an inch of new bone evenly distributed. Were this new bone formed from the contacting ends of the old bone of the stumps, it seems to me that it would shade off, becoming less the farther we went from the old bone. As a matter of fact, the greatest amount of new bone is at the fracture spot in the graft, which is at some distance from either contacting extremity of old bone. Fig. 4 K shows that the transplant (June 9, seven months after the grafting) has continued to develop. The upper part of the graft has become as large as the opposite healthy tibia. The fragments below the fracture, due to the inhibitory influence of the wire, have not well developed. In addition the wire is eroding the bone of the graft. I removed this wire June 14, 1913, with the object of preventing the cutting through of the graft.

Fig. 4 M shows that, six months after the transplantation, there is no filling in of the defect in the fibula with new bone since all the periosteum was removed with the graft. To have new bone fill up a defect it is absolutely necessary to leave either some bone or else some periosteum in place bridging the defect.

Fig. 4 P shows the result two months after the removal of the wire. The lower fragment which was being eroded by the wire has markedly developed and the fracture is consolidating nicely.

At the recent surgical congress in New York, Dr. Murphy said, "In bone transplantations there is but one absolute essential, other than rigid asepsis, and that is that the bone graft must be closely connected at some one point at least with living, freshened, osteogenetic tissue." From the preceding human transplantations, I judge that there is one other essential, and that is the presence of the periosteum.

ANIMAL EXPERIMENTS

Experiment 2. Fig. 6 shows the condition six months after the resection of the whole diameter of a rib together with its periosteum. The defect has not at all closed in. The periosteum was stripped bluntly from the bone section and this strip of periosteum was transplanted into the abdomen.

Result: A transverse section was cut six months later through this periosteum, which was felt to be bone. A photomicrograph of this section gives a picture of normal, healthy, living bone surrounded by a closely investing connective-tissue capsule. There are no osteoclasts present nor any round cells suggestive of either a destructive or an inflammatory process. I shall not go into a description of these microscopic sections but shall give you simply the results of them. This result shows that transplanted periosteum may produce living bone.

Experiment 3. This animal was killed eight months after the subperiosteal resection of a section of a rib (Fig. 7 A). This rib without periosteum was then split into longitudinal strips and these were transplanted into the abdomen.

Result: The defect in the rib from which the periosteum was not removed has become entirely filled in with new bone (Fig. 7 A). The strips transplanted into the abdomen have remained intact, have grown together, and have increased in size. A photomicrograph of a section of this abdominal transplanted bone shows bone perfectly alive, not undergoing absorption, and containing well-preserved and normal marrow. I attribute the living of these transplants to the fact that the splitting of the original rib allowed sufficient blood to get to the bone-cells.

This and the following experiment are refutations of Dr. Murphy's conclusion, in which he says that bone, with or without its periosteum, when transplanted into the soft parts and not in contact with living bone, always becomes absorbed.

Experiment 4. This dog was killed six months after subperiosteal resection of two ribs. The ribs without periosteum were broken up with the rongeur into the smallest possible fragments and these were transplanted into the abdomen.

Result: The defects made in the two ribs have acted differently one from the other (Fig. 8 A). The upper defect has about half filled in with new bone while the lower has formed a complete new rib, almost double its normal size. The fragments (Fig. 8 B) into the abdomen have lived, have increased in size, and have amalgamated into one homogeneous piece. A photomicrograph of a section, taken from these abdominal grafts, shows that the graft is perfectly alive, and contains normal marrow and normal blood-vessels. There are no osteoclasts present nor any signs of a destructive or degenerative process.

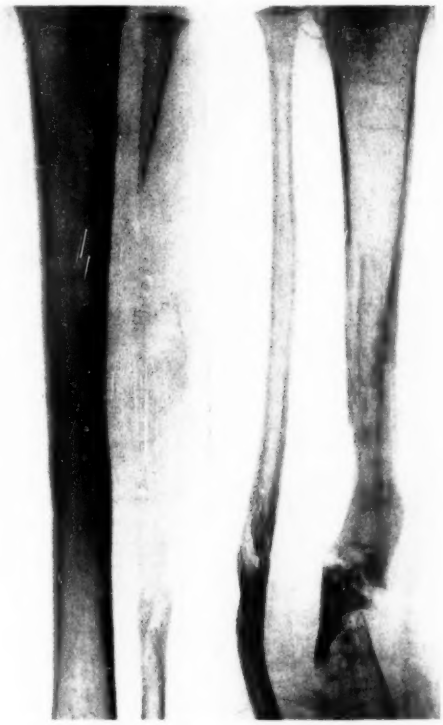

Fig. 4 M, Case 4. Fig. 4 P, Case 4.

The last two experiments clearly demonstrate that the life of a living graft depends entirely on its receiving sufficient blood to keep it alive, and nothing else. This was made possible by the splitting of the grafts which allowed sufficient blood to get to their cells. The next two pictures more clearly bring this point out, both being the same experiment.

Experiment 5. In this animal, killed after one month, two ribs were subperiosteally resected. One of the ribs was transplanted into the abdomen, into a defect made in the last rib, after the periosteum of the last rib was divided and separated but not removed. The second rib, also without its periosteum, was sutured to the end of the first rib, and both ribs in contact were buried in the abdomen.

Result: The first rib (Fig. 9 A) has healed in perfectly to the defect made in the last rib by a massive callus formed at the point of junction, evidently due to periosteal bony growth. There is no union or new bone formed whatsoever between the two transplanted ribs, owing, as I believe, to an

Fig. 6. Experiment 2. A, resected rib; B, periosteum with new formation of bone after six months, three weeks transplanted into the abdomen; C, transverse section for microscopical examination.

insufficient blood-supply to their cells and to the absence of periosteum to re-form the bone. In addition, the second rib shows beginning absorption, evidenced by its honeycombed appearance. The picture indicates that an entire graft on an entire graft, neither having any periosteum, will not live.

Experiment 6. This is the same experiment as the preceding. The animal was killed six months after four ribs were resected without their periosteum. One of the ribs without periosteum was sutured to a defect in the last rib, a second rib was sutured to the end of the first, and both ribs were buried in the abdomen. Fragments from another rib, which was divided into as small pieces as possible, were strewn along both the transplanted ribs.

Result: Fig. 10 shows that all the four defects created in the ribs have become completely filled in with new bone, owing undoubtedly to the presence of the periosteum, which had not been removed. The picture further shows that the second attached rib, as well as about half the first rib, have become entirely absorbed. Some of the fragments have become attached to the sides of the remaining part of the first rib.

The last four experiments are a convincing demonstration, it seems to me, of the fact that it is simply and solely a question of a sufficient blood-supply, which determines whether a graft will live or die, and not a question of whether the graft be in contact with living bone or not, as Dr. Murphy maintains.

Experiment 9. This animal was killed five months after one inch of the radius, together with its periosteum, was removed. The periosteum was then scraped off the fragment, which was split into as small pieces as possible with the rongeur. These small fragments were replaced, filling in the defect in the radius.

Result: The small fragments (Fig. 13) have all remained alive and have coalesced into one piece,

Fig. 7 A, Experiment 3. Middle of left rectus, sheath divided and strips laid on muscle, separated from each other, sheath sutured over these. Picture shows rib entirely reformed and growth together of bones in abdomen.

which has become united to the ends of the radius fragments.

This experiment illustrates the influence of a good blood-supply on grafts.

Experiment 12. This dog was killed twenty-four days after the periosteum was excised from a wide area of the humerus. Within this area was chiseled away a section of bone, extending into the medullary cavity, and the marrow and endosteum were thoroughly curetted away.

Result: Fig. 16 shows that the cavity made in the bone is irregularly filled in by new bone, the medullary cavity being quite obliterated by this newly formed bone. The periosteum can be seen ending at quite a distance from this cavity, and by no stretch of the imagination can one think that it had anything to do with the formation of new bone. Likewise with the endosteum. The new bone must then come from the bone-cortex itself, resulting from the efficient blood-supply from the nutrient artery. From these cases and experiments, we see that to have new bone fill in a defect it is necessary to have either some periosteum covering it or else at least a thin layer of bone bridging over the defect.

Experiment 13. The last statement made is exemplified in this animal, which was killed six months after the removal of an inch and a half from the radius, together with its periosteum. The animal was killed because he fractured the ulna, immediately opposite the defect in the radius.

Result: Fig. 17 shows that there has not been the slightest attempt to fill in the defect in the radius with new bone, owing, as I believe, to there being no periosteum left to produce it. If Macewen and Murphy are right in their views, why was this defect not filled in with bone coming from the ends of the fragments?

Experiment 14. This animal was killed three months after the removal of two inches of bone from two ribs, each with its corresponding periosteum. Each single fragment was transplanted into the defect made by the removal of the other fragment, each being completely covered by its periosteum. They were both held in place, end to end, by sutures

Fig. 8 A. Fig. 8 B.

Fig. 8 A, Experiment 4. Two ribs subperiosteally resected. Shows perfect reformation of one rib and imperfect reformation of other.

Fig. 8 B. A, section for microscopical examination; B, new bone.

passed through drill-holes in their extremities and through drill-holes in the ends of the fixed ribs.

Result: One end (Fig. 18) of each graft has not united to its corresponding extremity of the rib, owing to the respiratory motions. The other ends are solidly united to the fixed rib. In addition there is irregular production of new bone at various points on the grafts, due to periosteal growths. This result shows the great influence the periosteum exerts in preserving the life of grafts. Microscopic sections of each of these transplants show that they are both actively alive.

Experiment 15. This animal died six weeks after 2½ inches were resected from each fibula with the periosteum attached. Into the right fibula defect was transplanted the left fibula graft without any of its periosteum, the transplant being first divided into six fragments. Into the left fibula defect was transplanted the right fibula graft, which was previously divided into six fragments, on each of which was left its corresponding periosteum.

Result: The fragments without periosteum (Fig. 19) into the right fibula defect have become almost entirely absorbed. There is just a faint line indicating the little that remains of the grafts. In startling contrast to this is the result in the left leg, into which defect the fragments were transplanted with the periosteum on each fragment. These individual grafts have not been absorbed in the slightest; on the contrary, they have grown together and have increased in size. It is not possible to escape the conclusion that this good result in the left leg is entirely and absolutely due to the presence of the periosteum. If the result depended simply on the contact with the neighboring, living bone of the stumps, as Murphy maintains, why did not the fragments without periosteum in the right leg live?

Experiment 16. Fig. 20 shows the condition eight months after sections were taken from each fibula, from each of which the periosteum was carefully scraped. Into the left fibula defect was transplanted the entire, undivided, right fibula fragment without periosteum in contact with the ends of the living stumps. Into the right fibula defect was transplanted the left fibula fragment without periosteum, which was first divided into two longitudinal

Fig. 9 A, Experiment 5. The two transplants are shown, the upright or second transverse rib shows beginning absorption and also the absence of union between it and the adjacent transplant. The first transplanted rib shows an excessive callus and solid union due to periosteum.

strips, these being placed side by side in contact with the ends of the living stumps.

Result: The two fragments without periosteum (Fig. 20), comprising the graft into the right fibula defect, have lived perfectly and have united together and to the ends of the old stumps. In addition there is proliferation of new bone, uniting the graft to the neighboring tibia. In contrast to this is the result in the left defect. This graft without periosteum, transplanted entire, has very poorly united to the ends of the old stumps, and the transplant appears, from its poor shadow and irregular honeycombed appearance, to be absorbing. I attribute this difference in the result to the fact that the splitting of the graft allowed a much better blood-supply to reach the bone-cells of this graft than was possible in the case of the graft which was not split.

Experiment 17. Fig. 21 is very instructive and illuminating, and warrants close study. It was taken eight months after sections of bone were taken with the periosteum from each fibula. Into the left fibula defect was transplanted the right fibula graft, which was first broken up into the smallest possible pieces, the periosteum being thoroughly scraped from the small fragments. The pieces completely filled in the defect and were in contact with the ends of the stumps of the old bone. Into the right fibula defect were transplanted fragments of the left fibula graft with periosteum still attached to each small graft.

Result: Fig. 21 indicates the great value of having periosteum on grafts. The small fragments without periosteum in the left defect have become largely absorbed, being much reduced in number. Those which remain are isolated, are not united, and appear to be absorbing. In marked contrast to these are the fragments transplanted into the right defect with periosteum. They have become united to each other in most instances, have increased in size and produce the deep shadows of healthy, living bone.

This experiment is another instance of the fact that contact with living bone does not

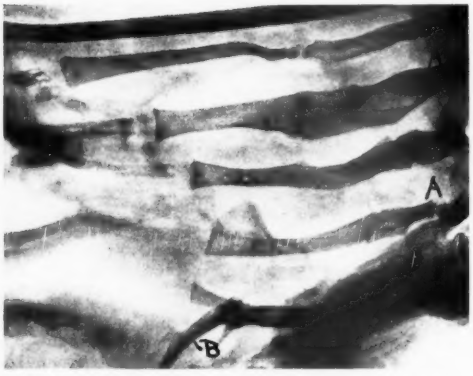

Fig. 10. Experiment 6. A, four resected ribs all filled in. Removed without periosteum. B, transplanted rib all that is left of two grafts. Section from this spot for microscopic examination.

seem to be the important element which Dr. Murphy has attributed to it. This experiment is a further proof of the statement, emphasized by me, that without periosteum there is always uncertainty as to whether grafts, even though they be small, will acquire enough blood to keep them alive. With the periosteum on the grafts one is sure of their living, I believe, as the result of one or two things happening: either a sufficient blood-supply, or a resupply to the grafts of living cells, derived from the periosteum, which reproduce the bone.

Experiment 18. A transplantation, in which, into a defect made in the left fibula, were transplanted fragments obtained from the right fibula, each covered with its periosteum. Into the right fibula defect were transplanted fragments from the left fibula section, from which all the periosteum was first carefully scraped away.

Result: The result in this dog confirmed the view that the life of grafts depends simply and solely on the blood-supply which they obtain. The fragments with periosteum into the left fibula defect appear to have lived perfectly, to have increased in size, and to have solidly united with each other and to the ends of the old, living stumps. The same process occurred on the right side, into which defect fragments without periosteum were transplanted.

Experiment 19. In this experiment into a defect made in the left fibula were grafted fragments, taken from a section of the right fibula, from which all the periosteum was first carefully scraped away. Into the defect made in the right fibula were transplanted fragments with periosteum on each piece, taken from a section of the left fibula.

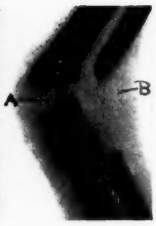

Fig. 13. Fig. 16. Fig 17.

Fig. 13, Experiment 9. A, pieces of graft.

Fig. 16, Experiment 12. A, edge of periosteum; B, new bone.

Fig. 17, Experiment 13. A, fracture; B, defect in bone not filled in because of removal of periosteum with the bone.

Result: The fragments transplanted without periosteum into the left fibular defect have almost completely disappeared. The fragments transplanted into the right defect with periosteum have remained living, have markedly increased in size, and have largely united to each other and to the ends of the old stumps. The feature to be particularly noticed in this picture is the pronounced increase in size of the right-sided grafts, denoting the active life of the transplants, and also the fact that, although the middle section of the graft on the right side does not seem firmly united to the neighboring transplants, yet this middle section appears to be as actively alive as the sections which are in intimate contact with the ends of the old stumps; all of which goes to show that contact with living, old bone has not the influence on the subsequent life of grafts that Dr. Murphy has attributed to it. Far more, as I have repeatedly tried to emphasize, do I believe that it is solely a question of sufficient blood getting into the graft.

The accompanying two tables give the results obtained in all of these transplantations.

From Table I it may be seen that 93 per cent of all transplantations made with the periosteum were successful, while but 48 per cent of those made without the periosteum were successful.

TABLE I.—RESULTS OF GRAFTS WITH AND WITHOUT PERIOSTEUM

			Totals	
With—	Animal	Human	No.	Per Cent.
Successful	13	1	14	93.3
Unsuccessful	1	0	1	6.7
Without—				
Successful	12	0	12	48
Unsuccessful	9	4	13	52

TABLE II.—GRAFTS WITHOUT PERIOSTEUM: RESULTS OBTAINED IN TRANSPLANTATIONS WITHOUT THE PERIOSTEUM ACCORDING TO THE SIZE OF THE GRAFTS

			Totals	
Small fragments —	Animal	Human	No.	Per Cent.
Successful	7	0	7	50
Unsuccessful	7	0	7	50
Larger, single grafts —				
Successful	5	0	5	41
Unsuccessful	3	4	7	59

From Table I it is seen that 48 per cent of all the transplantations made without periosteum were successful. From this I reason that there must be some other factor present, making for the success of these grafts, than the mere covering of the periosteum, and this I infer must be the presence of a sufficient blood-supply to keep them alive. Had the periosteum been on the grafts, it may be safely said that they would practically all have survived. Just exactly what the influence is which the periosteum exerts, it seems difficult to state positively. Whether its presence affords a better blood-supply to the grafts, or whether the periosteum, in the event that the bone-cells in grafts have died, supplies fresh cells to the grafts and from these regeneration takes place, must for the present remain unsettled. That the blood-supply is more than likely the important point is seen from Table II. It goes without saying that a small fragment will get a sufficient blood-supply to its cells more readily than a single, large piece, hence is more likely to remain living. This important fact is proved in Table II, in which it is shown that 50 per cent of the transplantations in which small fragments without periosteum were grafted were successful, while but 41 per cent of the transplantations in which single, larger pieces without periosteum were grafted were successful.

I think, in conclusion, that I have demonstrated that both Macewen and Murphy are mistaken in their conception of the lack of function of the periosteum in maintaining the life of grafts. It seems to me likewise that Murphy is misled in his conception that the graft is not osteogenetic in itself. This fact is clearly seen in Experiments 3, 4, 17 and 19. He says that the graft is simply osteoconductive of cells into the graft from the contacting extremities of the neighboring old bone. If this be the case, why should so many of the grafts without periosteum have died in which this contact was thoroughly carried out?

CONCLUSIONS

The conclusions from the preceding human transplantations and animal experiments are:

1. If the periosteum be excised from an area of bone and a section of bone within this area, going into the medullary cavity, be removed (not, however, involving the whole diameter of the bone) and the endosteum scraped away as well, then it is found that the cavity fills in perfectly with new bone, which of necessity must come from the old bone alone. This is probably due to the fact that the nutrient artery is intact.

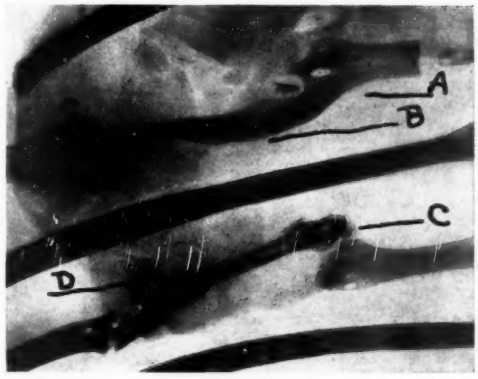

Fig. 18, Experiment 14. A, solid union here; B, section taken for microscopic examination; C, no union due to respiratory motions of chest, notice periosteal outgrowths; D, section taken for microscopic examination. Union here.

Proofs: Human transplantation, Case 4, in which the defect in the tibia filled in perfectly; Animal Experiment 12.

2. If a section of the whole diameter of a bone be removed, then the bone will regenerate between the ends of the fragments if the whole or a part of the periosteum be preserved bridging the defect.

Proofs: Animal Experiments 3, 4 and 6; Bashkirzew's human case (see my Conclusion 6). The subsequent reformation of the ribs after human empyema operations performed subperiosteally.

3. If a section of the whole diameter of a bone be removed, very little subsequent filling in of this defect by bone will take place if the entire periosteum has been removed from between the ends of the fragments.

Proofs: Animal Experiments 1, 2, 13, 15 (right), 17 (left), 19 (left) and 20 (left). Human transplantation, Case 4, in which a great part of the fibula with all its periosteum was removed. There is very little of this shaft which has been reformed. In case that we wish to have a bone reform, it seems necessary to leave some of its periosteum in position.

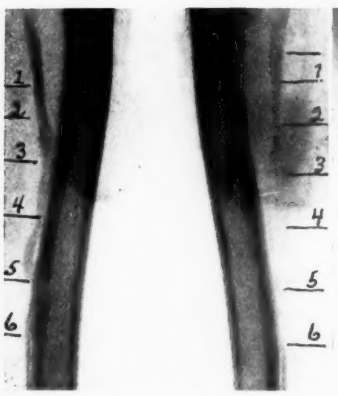

Fig. 19, Experiment 15. Points of section.

Fig. 20, Experiment 16.

Fig. 21, Experiment 17.

This is brought out by Frangenheim[1] and von Hacker.[2] They each took pieces from the fibula, comprising the whole thickness of the bone together with all the periosteum covering these fragments. There was no subsequent reformation of bone in the defects. Frangenheim consequently recommends that in taking a section from the fibula, a small strip of periosteum, from which the fibula will be reformed, be left on the interosseous membrane.

4. Provided a graft be living and taken from the same patient, its future life depends on an efficient blood-supply, irrespective of the periosteum or whether it is in contact with living bone or not. This conclusion I wish to emphasize strongly.

Proofs: Animal Experiments 3, 4, 8, 16 and 18.

5. If minute fragments of a living graft be transplanted, then the periosteum may be disregarded because the osteoblasts in the fragments may not die because of an easy access of blood into them, and the pieces may grow and coalesce and not become absorbed. My statistics show that 50 per cent of such transplantations will be successful.

Proofs: Animal Experiments 7, 9, 16 and 18, also Macewen's human case. Thus, in his book he narrates the case of a boy, the whole of the diaphysis of whose humerus he was compelled to remove for necrosis. There was no subsequent osseous deposition. Fifteen months later, two wedges of bone were excised from another patient 6 years of age, affected with anterior curves. These were cut into minute fragments, quite irrespective of the periosteum, and were then deposited in the muscular sulcus in the boy's arm. Two months later, a portion of new bone, an inch in length and three-quarters of an inch in thickness, was found firmly attached to the upper fragment of the humerus. Here all the grafts proliferated, grew to one another and also to the extremity of the proximal portion. Two other wedges of bone of larger size than the first and broken up into fragments were similarly dealt with and inserted two months subsequently to the first graft, and a third couple were placed in position five months after the first. These all fused together and to the condyles of the humerus and filled the gap in the arm to the extent of 4 inches. It is now thirty years since the shaft of the humerus was rebuilt, and, during that period, the man has depended on his physical exertions for his livelihood. He is now an engineer's pattern-maker. Other cases in which minute fragments of bone have been successfully transplanted are on record. Thus, Vallas[3] reported the case of a woman on whom he had operated five months previously for a pseudo-arthrosis of the tibia. At the operation, he found that the ends of the fragments touched at only a small point, due to the slender form of the inferior fragment. He then removed from the rounded extremity of the upper segment ten or twelve bony chips which he placed in the defect between the bones. The periosteum and soft parts were then sutured about these chips. Primary union resulted and in six weeks consolidation was complete and the patient left the hospital. Barth[4] reports two successful cases in each of which he transplanted two fragments, taken from the tibiæ, each covered by periosteum. In both of these cases, the fragments grew and proliferated and filled in the defects perfectly. Even though one is safer in transplanting small fragments without the periosteum than larger ones (compare Experiments Nos. 16 and 18), at the same time the

[1] Fragenheim: Arch. f. klin. Chir., 1909, xc, 438.

[2] Von Hacker: Quoted by Streissler, Beitr. z. klin. Chir., 1910, lxxi, Part 1.

[3] Vallas: Lyon méd., 1894, lxxvii, 606.

[4] Barth: Arch. f. klin. Chir., 1908, lxxxvi, 860.

small fragments may die if they are without the periosteum (compare Experiments Nos. 15, 17, 19 and 20).

6. If a larger piece of living bone be transplanted, then it is safer to leave the periosteum attached to the graft to assure its future reformation, since otherwise the osteoblasts in the bone of the graft may die because cut off from their nutrient artery. The periosteum survives because of its sufficient blood-supply from the surrounding tissues, its inner surface forms osteoblasts which themselves proceed to reform the bone of the graft, in the event that the osteoblasts within this have died from a deficient blood-supply. My statistics show that 41 per cent of transplantations are successful in which large grafts without periosteum are grafted.

Proofs: Previous Cases (human) 1, 2, 3 and 4; Animal Experiment 14. For the sake of being explicit, I wish to state that this conclusion does not imply that the periosteum is unconditionally necessary for the growth of transplanted bone. It merely asserts that one is much surer of the grafts living if the periosteum is present on the graft. There are comparatively few cases on record of grafts having been made without the periosteum. I presume that this is due to the fact that it is not customary to report non-successes. I have come across the following two cases which were successful after transplanting grafts without the periosteum: Baskirzew[1] reports a case which illustrates two things; first, how well the defect in a bone made by removing its entire diameter will fill in, provided the periosteum be left; and, second, how well a graft may live and grow at times even when transplanted without its periosteum. Thus, he removed a 9 cm. piece without its attached periosteum, from the diaphysis of a fibula for the purpose of a transplantation. Four years afterward, in the place where the fibula was removed, a stout, although uneven, fibula resulted. The piece from this fibula was transplanted in a defect made in the ulna for the removal of a supposed sarcoma in which not only the bone but also its periosteum was entirely removed. The graft in the ulna without periosteum, transplanted into a bed not lined by periosteum, grew in perfectly and consolidated with the upper fragment, a fact due, as I believe, to sufficient blood having been supplied. The second case is that by Wetherill.[2] On September 28, 1912, he grafted a segment, three and a third inches long, from the opposite tibia into a defect in the tibia resulting from osteomyelitis. The graft was taken without its periosteum. A röntgenogram taken after six months shows that the graft has lived and has increased n size.

7. A graft on a graft, neither having a covering periosteum, will not live, even though one graft be in contact with living bone.

Proofs: Animal Experiments 5 and 6.

8. Periosteum when transplanted alone into the soft parts may produce living bone.

Proof: Animal Experiment 2. The remarkable vitality of transplanted periosteum has been demonstrated by Grohe[3] and Morpurgo,[4] the former showing that it is capable of preservation for one hundred hours and yet able to be implanted and preserve its osteogenetic powers; the latter showing that the periosteum of a corpse, kept at 15° C., can produce new bone when transplanted after 168 hours.

9. The splitting of the periosteum of a graft, even though the transplant is entirely surrounded on all sides by periosteum, seems to be unneccessary and accomplishes nothing.

Proof: Previous Case 4 (human), in which the fibula graft was transplanted with periosteum on all its sides and this wâs unsplit. The graft grew beautifully.

10. Altogether sixteen transplantations with periosteum were performed and of these all but one (93 per cent) were successful.

11. Altogether twenty-five transplantations without periosteum were performed. Of these 48 per cent were successful (all in animals), while 52 per cent were unsuccessful. The influence of the blood-supply is demonstrated by the fact that 50 per cent of the transplantations without periosteum, made with minute fragments, were successful, while 41 per cent of those without periosteum, made with large, single fragments, were successful.

12. From a consideration of all the foregoing facts, the conclusion seems irresistible that bone-grafts of whatever size, in order that one should be positively assured of their subsequent living, should be transplanted with as much periosteum covering their surfaces as is possible.

I desire to thank Dr. Joseph Blake for the privileges of the laboratory, Dr. William C. Clark for the pathologic examinations, and Mr. Ira Simms, of the Presbyterian Hospital, for the excellent röntgenograms.

[1] Baskirzew: Deutsche Ztschr. f. Chir., 1912, cxiii, 524.

[2] Wetherill, H. G.: The Growth, the Death and the Regeneration of Bone. J. Am. M. Ass., 1913, March 29, p. 983.

[3] Grohe: Virchow's Arch. f. path. Anat., etc., Berl., 1899, clv, 428.

[4] Morpurgo: Virchow's Arch. f. path. Anat., etc., Berl., 1899, clvii, 172.

THE SURGICAL TREATMENT OF INTRAMEDULLARY AFFECTIONS OF THE SPINAL CORD[1]

By CHARLES A. ELSBERG, M. D., New York City

With the increase in our knowledge of diseases of the spinal cord and its membranes, and the advance in our surgical methods, operations for affections of the cord have become increasingly frequent. Up to within the last few years, however, surgical interference in disease of the substance of the spinal cord was rarely, if ever, attempted. Seldom had anyone ventured to invade the delicate tissues of the cord, for fear of causing greater damage than that produced by the disease. Excepting in rare instances, such as Abbe's (1) attempt to empty a hydromelia cavity by aspiration, intramedullary cord affections were considered hopeless. If the diagnosis of intramedullary disease was made, operative interference was considered contra-indicated; if an affection of the cord itself was found at operation, the wound was closed without delay.

It was perhaps fortunate for the progress of spinal surgery that the differential diagnosis between intramedullary and extramedullary disease was often uncertain, that such signs as absence of pain and the dissociation of sensations were not always sufficient to permit a definite conclusion. Hence intramedullary affections were encountered in the course of spinal operations and the surgeon gained a better knowledge of the physical character of intramedullary spinal disease. Then followed isolated instances in which the surgeon ventured to invade the cord substance—Krause (2), Röpke (3), von Eiselsberg (4), Veraguth and Brun (5), Braun (6), Schultze (7), Elsberg and Beer (8), etc. These surgeons were cognizant of the dangers of manipulation of the cord, of the irreparable damage that might result from the slightest trauma, and the danger of transverse lesions from injury to the cord tissue; that in spite of this they ventured upon this unexplored field is another evidence of the restless forward march of scientific surgery.

All these efforts to attempt the surgical relief of affections within the substance of the cord were, however, only isolated efforts; they were not based upon regular, well worked out methods of procedure: they lacked a carefully worked out anatomical foundation and the necessary special technique.

During the past three years, I have made a study of the anatomy and physiology of the spinal cord from the viewpoint of intramedullary surgery, have attempted to develop a special technique, and have applied this technique to the operative treatment of some intramedullary diseases. The results of these investigations and experiences are herewith described.

THE FREQUENCY OF INTRAMEDULLARY SPINAL DISEASES WHICH MIGHT BE SUBJECTED TO SURGICAL TREATMENT

Intramedullary tumors are not by any means as rare as they are believed to be by some authors. Thus Allen Starr (9), quoting from Schlesinger, states that among 302 tumors of the cord, 125 were within the cord substance. These included sarcoma, tuberculous tumors, gumma, glioma, lipoma, cysticercus, neuroma, cholesteatoma, myxoma, teratoma, adenoma, endothelioma, etc. Many of these intramedullary growths infiltrate the cord substance and increase in size by extending upward and downward, but some enlarge in their transverse diameter, are encapsulated and of small size. Gowers (10) says that "these tumors in some cases blend with the substance of the cord, but in other instances they are bounded by an area of softening which often isolates even invading growths." According to Flatau and Gowers, many tumors are sharply limited. Flatau (11) states that the comparative frequency of extramedullary and intramedullary tumors is as 2.6 to 1 (57 intramedullary tumors in 213 cases). Although one cannot estimate from the reports the frequency of localized and

[1] Read before the Section on Neuropathology of the XVIIth International Congress of Medicine, London, 1913.

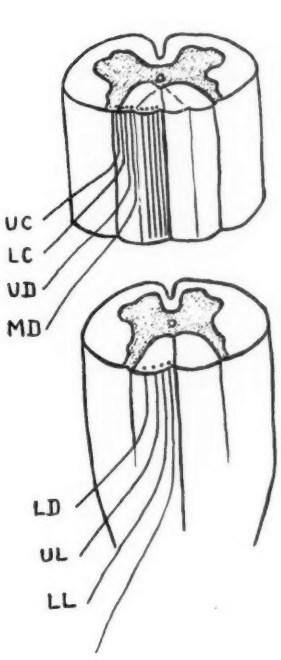

Fig. 1.

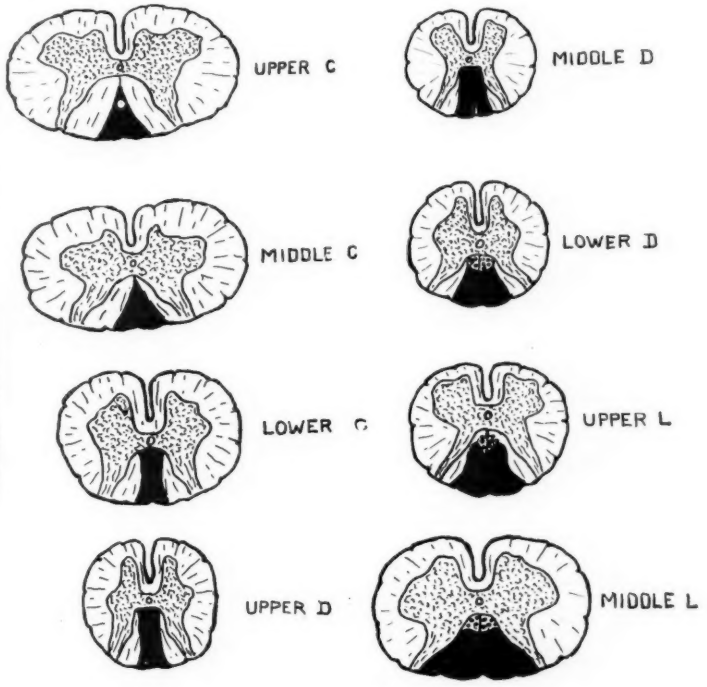

Fig. 2.

Fig. 1. The conducting paths in the posterior columns of the spinal cord, showing that the fibers from the sacral (S) and the lower lumbar (LL) posterior roots lie nearest the posterior median fissure (diagrammatic).

Fig. 2. The areas in the posterior columns in which ascending degeneration occurs after injury to the sacral and lower lumbar posterior roots of both sides (after Schultze). Within these areas, shaded in black, an incision can be made which will cause little injury and few, if any, symptoms.

encapsulated intramedullary growths, the fact that one frequently finds illustrations and descriptions of such tumors in the literature is evidence for the contention that they are not so very rare. Localized tumors have been found at operation by Krause, Röpke, Elsberg and Beer, and many others. Localized cysts have been found by Krause and myself. That localized collections of fluid occur in syringomyelia and hæmatomyelia quite often is well known. Foreign bodies have been found lodged in the cord substance. In the instances cited, the affection of the cord is of such a nature that, physically at least, its removal or eradication should be possible.

Aside from these more strictly localized affections, there are others of a more diffuse nature in which surgical interference might be of benefit. Thus the drainage of a hæmatomyelia or hydromyelia into the subdural space, if it were possible of accomplishment with safety, should at least remove the harmful effects of the pressure of the fluid upon the nerve fibers in the cord. If an internal decompression of the cord were possible, the harmful effects of slow growing and infiltrating tumors should be decreased by an incision into the cord, as should also be the œdema which follows trauma or contusion of the spinal cord after injury to the spine (Allen). A decompressive incision into the cord might also do some good in spinal gliosis.

Isolated cases are to be found in the literature of the subject in which intramedullary tumors were removed with great improvement

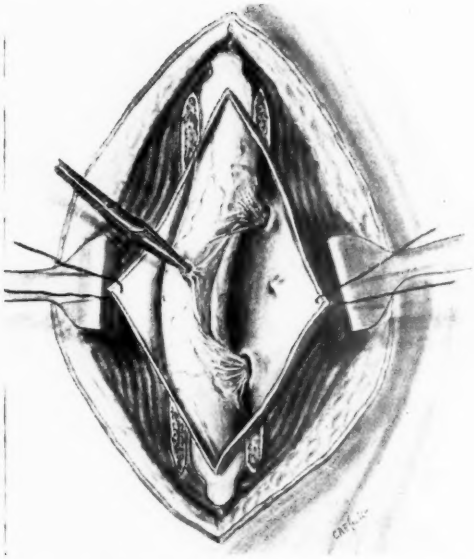

Fig. 3. The method for the exposure of the anterior surface of the cord. The cord is rotated by means of traction on a divided slip of the dentate ligament.

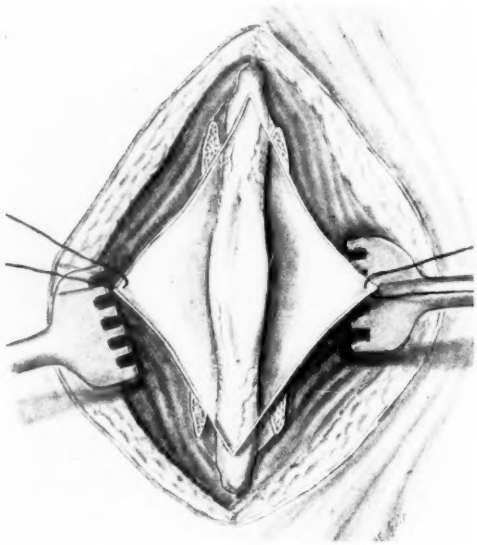

Fig. 5. Appearance of cord after incision of hydromyelia cavity. The openings of the central canal can be plainly seen.

in the patient's symptoms—Röpke (3), von Eiselsberg (4), Schultze (7), Elsberg and Beer (8); intramedullary cysts and foci of softening have been drained—Krause (2), Elsberg (8); a small, well encapsulated tubercular focus has been removed—Veraguth and Brun (5); the attempt has been made (Abbe, Krause, Elsberg) to drain a syringomyelitic collection of fluid into the subdural space. The cord has been incised in order to remove a bullet from its substance—Braun (6)—and for the purpose of decompression in extensive irremovable tumors (Cushing, Krause, Elsberg).

Fig. 4. The appearance of a subpial spinal tumor. Tumors in this location have sometimes been mistaken for true intramedullary neoplasms.

THE SURGICAL ANATOMY OF THE SPINAL CORD FROM THE STANDPOINT OF INTRAMEDULLARY SURGERY

Not only from their anatomical position and their physiological character, but also from the ease with which they may be exposed, the posterior columns of the spinal cord are the most favorable for an incision. The fibers in the posterior columns convey principally tactile and deep muscle sensations to higher levels, although the fibers for all varieties of sensation enter the cord in the posterior spinal roots. Our knowledge of the

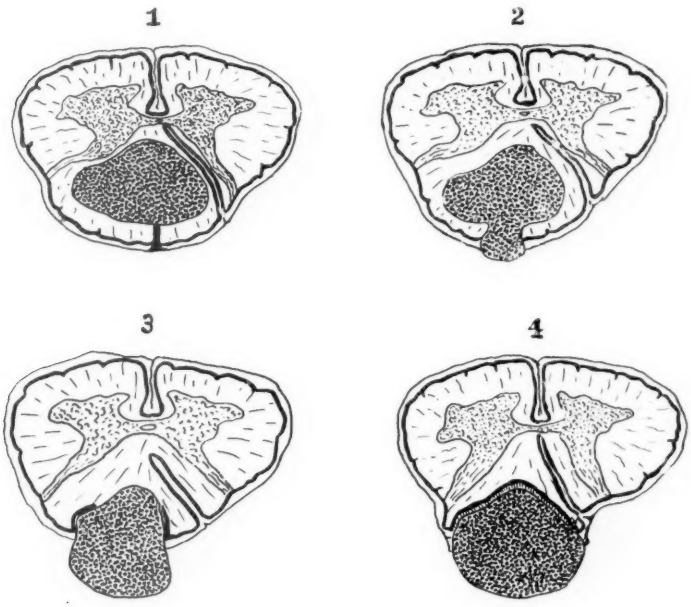

Fig. 6. The extrusion of an intramedullary tumor after the incision into the cord has been made (diagrammatic).

conducting paths for the various sensations is still incomplete; the following is a summary of all that is known:

The fibers for the sensation of pain enter the cord in the posterior roots, and soon pass through the posterior horn and gray matter to the anterior part of the lateral column on the opposite side. The fibers for thermæsthesia take a similar course; they do not cross to the other side so quickly as the pain fibers, but require from two to five segments for the complete crossing. The fibers for touch pass partly along the same course as the pain fibers and partly up the posterior columns on the same side to the posterior column nuclei at the extreme upper end of the cord. In these cloumns also lie the paths for deep muscle sense (postural recognition, spacial discrimination, etc.). When one part of the sensory pathway in the cord is interrupted, other tracts may take up the transmission of sensations. Although this is probably true for all kinds of sensation, the tactile sensations especially seem to run in a bilateral path and are not so apt to be markedly affected by a purely unilateral lesion.[1]

The posterior columns, in short, contain mainly fibers (endogenous and exogenous, primary and secondary paths) which contribute to the transmission of tactile and deep muscle (kinæsthetic) sensations, and we must next determine the course of these tracts in the posterior columns.

As is well known, the fibers from the posterior roots from below upward enter the cord and lie at first just mesial to the posterior gray horn. Some of these fibers remain in the posterior column, where they divide into ascending and descending branches. The ascending branches give off numerous collaterals to the gray horns until the level of the next root above is reached. Fibers from this second root then take their position on the mesial side of the posterior horn, crowding the fibers from the preceding (lower) root more toward the median line. It will be seen,

[1] Rothmann believes that sensory fibers for touch exist also in the anterior columns of the cord.

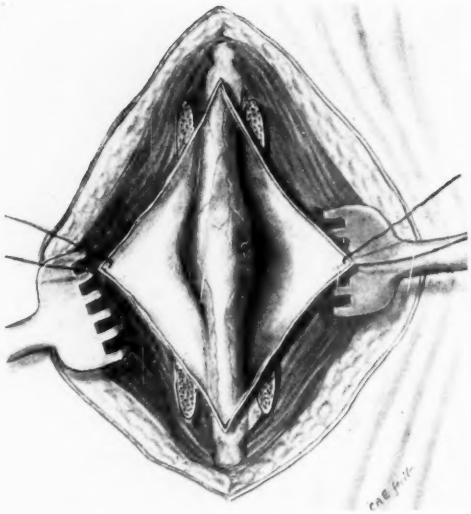

Fig. 7. Case 1. The fusiform enlargement of the cord due to intramedullary tumor.

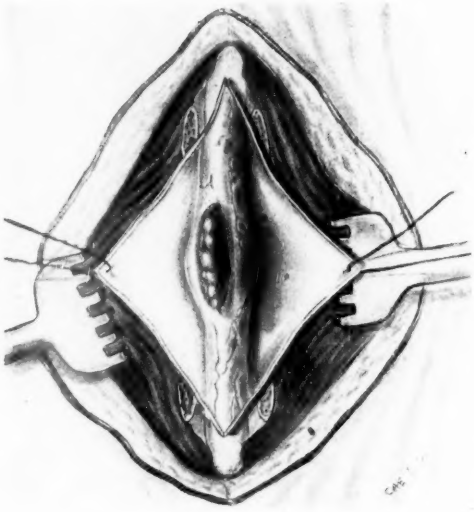

Fig. 9. Case 1. The complete extrusion of the tumor one week after incision of the cord. The tumor lies outside of the cord.

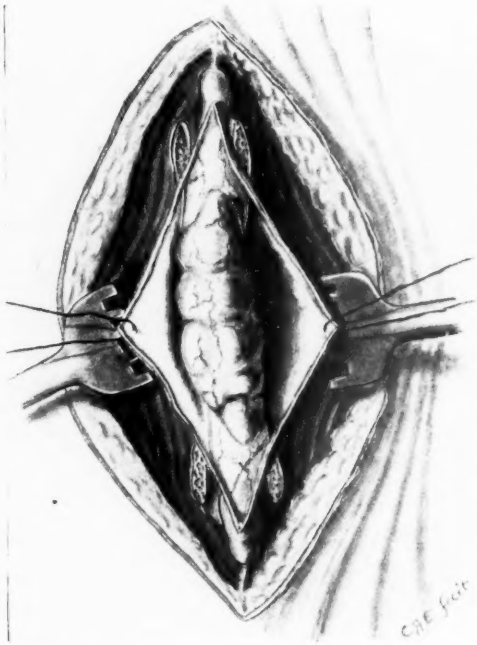

Fig. 8. Partial extrusion of the intramedullary growth in Case 1.

therefore, that the fibers finally form a succession of lamellar tracts which in each case lie at first next to the posterior horn and gradually become shifted medianward by those which enter the cord with the higher nerve roots (Schaefer). This arrangement of tracts in the dorsal columns has been studied in the lower animals by Kahler and many others, while degeneration in the tracts of the cord in man have been described in detail by Schaffer, Ziehen, Collier and Buzzard, Wickmann, Schultze, Petren, and others.

As a result of this arrangement of conducting paths in the posterior columns, the fibers from the sacral posterior roots soon come to lie nearest the posterior median fissure; outside of these run the lumbar root fibers, outside of these, the dorsal, and so on (Fig. 1).

In the lumbosacral region, the columns of Goll and Burdach are not distinct, and the fibers from the posterior roots occupy a great part of the dorsal column; while in the cervical and dorsal regions, the arrangement is such that the posterior median column (Goll) contains only fibers from the sacral, lumbar, and lowest dorsal posterior nerve roots, while the postero-external column of Burdach con-

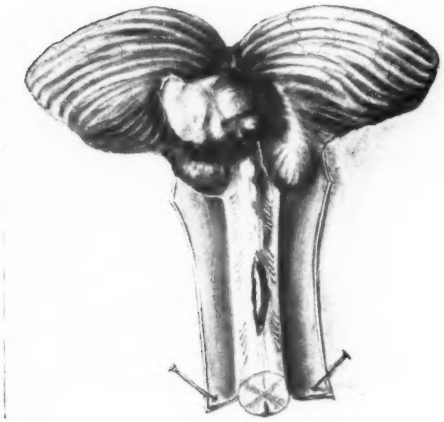

Fig. 10. Drawing from post-mortem specimen of an intramedullary spinal tumor which has been slightly extruded. The growth extended into the medulla and had grown into the posterior fossa. Extrusion of the spinal growth was impossible on account of the tumor mass above. The growth was at the level of the second cervical segment, and death was due to respiratory paralysis two days after the first operation.

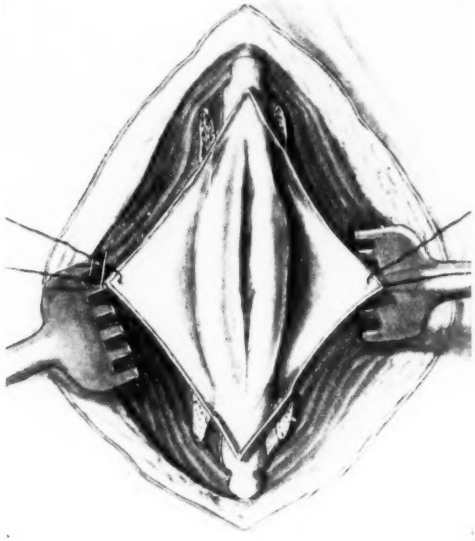

Fig. 11. The appearance of the cord in Case 7 after incision of the swollen cord.

tains the fibers from the upper dorsal and cervical posterior roots (Fig. 1). Therefore, in the lumbosacral region, an incision may be made anywhere in the posterior column. It is best made a few millimeters away from the median line, so that injury may not be done to the descending tracts (triangle of Gombault and Philippe, from the third sacral nerve to the conus) in the sacral region; the oval bundle of Flechsig (from the third and fourth lumbar nerves to the third sacral nerve), and the septomarginal bundle of Bruce and Muir (from the first to the third lumbar nerves) in the lumbar region. The incision should not be too near the posterior root zone, lest it do damage to the marginal fibers. The deeper the incision, the greater the probability of injury to fibers from the higher lumbar roots.

In the dorsal and cervical regions, the incision should always be made in the posterior median column, and the higher the level, the nearer should the incision be to the median line. In the upper and middorsal regions the incision is best made from 2 to 4 mm. from the median line, while from the level of the midcervical region upward it should be

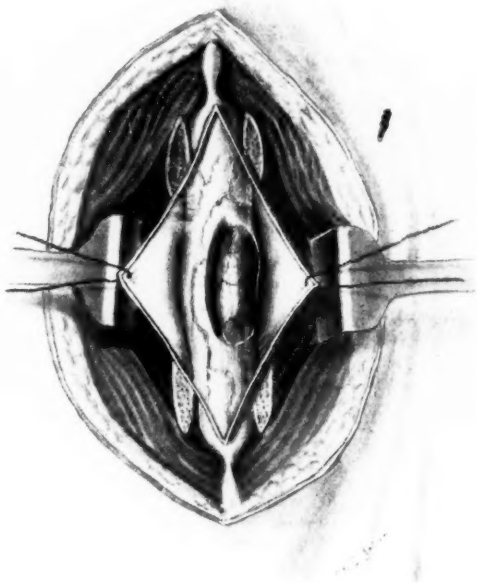

Fig. 12. An extramedullary tumor which lies embedded in the cord. This type of growth has been mistaken for an intramedullary growth.

Fig. 13. Case 2. The enlarged cord due to an intramedullary tumor.

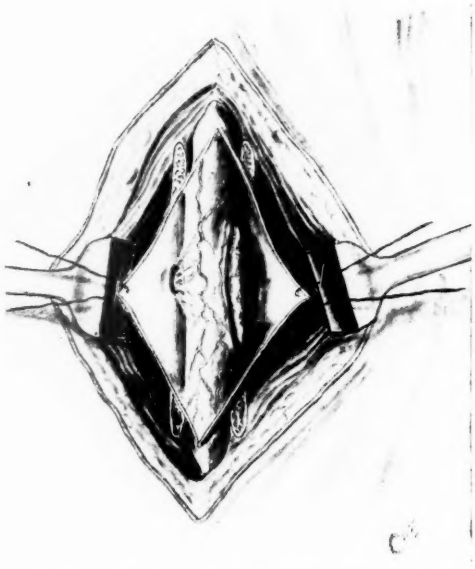

Fig. 14. Case 2. The appearance one week after incision of the cord. The tumor, a fibrocyst, has been extruded and lies on the side of the cord.

made very near the median line. The accompanying diagram (Fig. 2) shows the areas in the posterior columns of the human spinal cord in which ascending degeneration occurs after injury to the sacral and lowermost lumbar posterior roots of both sides. Within the areas shaded in black, an incision, made in the posterior column of the one or the other side, will cause a minimum of injury to nerve fibers, and few or no symptoms.

It is important also to remember that in the sacral region there is very little white matter, the bulk of the cord being formed of gray matter. In the thoracic region there is a relatively small amount of gray matter, while in the lumbar and cervical enlargements the gray matter, as well as the white columns, are of large size.

THE TECHNIQUE OF ASPIRATION AND INCISION OF THE CORD

The cord can be punctured with a needle with entire safety, provided that only the finest aspirating needle is used, and care is taken not to injure even the smallest vessels which enter the cord through the posterior median septum or run in the pia mater. In doing the aspiration, it is advisable to make the puncture near but not through the posterior median septum. The cord must be carefully steadied, as the pia offers some resistance to the entering needle. A slip of the dentate ligament is grasped on each side with fine mosquito forceps and held in place. Extreme care must be taken that the direction of the needle is held constant. Needless to add, the cord must never be grasped with an instrument or with the fingers.

For the incision of the cord, a very fine scalpel, such as the von Graefe knife used by the ophthalmologist, should be used. The arachnoid is first incised and grasped with fine forceps. Then the proper part of the posterior column is selected and an incision less than 0.5 cm. long is made. The incision must be carefully deepened and enlarged, care being taken that it shall be in the long axis of the cord. The enlarging and deepening of the primary incision is best done with a blunt instrument. A small strabismus hook is useful; by its use fewer fibers are divided and more are separated from each other.

The incision should always be slowly made, and should be stopped as soon as there is the slightest bleeding. Sponging should be done with small bits of cotton, with extreme gentleness, so as to make the smallest amount of pressure upon the cord itself. When a cystic collection of fluid or a small area of softening is opened, no effort should be made to remove the fluid in the cavity by sponging or by pressure.[1]

THE EXPOSURE AND RECOGNITION OF INTRAMEDULLARY LESIONS

A complete laminectomy is always necessary when a thorough exploration and examination of the cord is to be made. This usually means that at least three spinous processes and laminæ have to be removed, and that the laminæ shall be taken away well out to their articulating surfaces. While the operation of hemilaminectomy has its field of usefulness, a wider exposure of the cord is necessary whenever a tumor has to be removed or the cord tissue has to be invaded.

After the dura has been incised, the cord is exposed covered by the pia and the arachnoid, which is usually raised from the cord by fluid. The arachnoid should not be injured when the dural sac is opened and should be incised separately.

It should never be difficult to recognize a tumor that lies underneath the arachnoid membrane and outside of the cord tissue and pia mater, for these growths always lie superficial to the dentate ligament. It is not so easy, however, to differentiate between a subpial growth closely adherent to the cord (see Figs. 4 and 12) and a true intramedullary neoplasm, especially if an intramedullary tumor has broken through the posterior surface of the cord and has then pushed the pia before it. After the surgeon has incised the pia covering the tumor he may think that he is dividing cord tissue when in reality he is incising the capsule of the tumor. At some of my intramedullary operations, I have not been able to convince some colleagues of the true intraspinal location of the lesion because they were not sufficiently acquainted with the appearance of divided cord tissue. In several of my early operations, I was myself in doubt until I had had microscopical examinations made of post-mortem specimens, and of small bits of the tissue which lay superficial to the growth and which was incised before the growth was exposed. In the cases of intramedullary cyst formation, I was able to see the central canal of the cord which opened into the cyst cavity (Fig. 5).

With increasing experience, one is able to clearly recognize when the soft, creamy yellow cord tissue is divided; not only is its color different from that of the pia and of a capsule of a new growth, but also its consistency is very characteristic.

Careful palpation of the cord will often inform the operator that a solid tumor exists in the cord or that the enlargement is due to a collection of fluid. The majority of intramedullary tumors cause a fusiform enlargement of the cord (Fig. 7), so that, as soon as the dural sac is open, it is evident that the lesion is inside of the spinal marrow.

THE REMOVAL OF INTRAMEDULLARY TUMORS OF THE SPINAL CORD BY THE METHOD OF EXTRUSION

The attempt to remove a growth from within the substance of the spinal cord, no matter how carefully it was done, would require an amount of handling and a degree of manipulation of the delicate tissues of the cord that would, in the large majority of cases at least, be certain to be followed by a traumatic inflammation that would damage or destroy all the elements of the cord at the spot and would probably be permanent in its effects. Therefore, it was necessary to devise a new method for these intramedullary new-growths, and two years ago, together with Dr. Beer, I described a method of operation to which I gave the name "the method of extrusion," by means of which it is possible to remove a tumor from the substance of the cord with a minimum amount of injury to the cord tissue (Figs. 1, 2, 3, 4, and 6).

The theory upon which the method of extru-

[1] The cord may be raised from its bed by means of traction on a posterior root or on a slip of the dentate ligament. If the anterior surface of the cord has to be exposed, this can be satisfactorily accomplished by the division of one of the slips of the dentate ligament, and traction and rotation of the cord by means of the forceps which grasps the divided slip (Fig. 3). The importance of the dentate ligament is very great and its exact structure and relations should be carefully studied by everyone interested in spinal surgery.

sion is based is the following: There must exist within the cord a certain intramedullary pressure, as exists in all solid or fluid substances. When a tumor grows in a part of the cord, the intramedullary pressure must be locally increased. If an incision is made down to the growth, nature will proceed to readjust the pressure conditions, and in so doing, will push out—the conditions being favorable—whatever has caused the local increase of pressure. When, therefore, an incision is made down to the tumor in the cord, the tumor will be slowly pushed out of its bed and will be slowly extruded from the cord substance. Nature will accomplish this process slowly—nerve fibers being slowly pushed to the sides while the growth is being extruded—and with far less injury to the cord structure than by the most careful manipulations of the surgeon.

If these theoretical considerations were correct, then the surgeon would only have to incise the cord down to the tumor and then have to wait for the readjustment of pressure conditions when the tumor should be found outside of the cord tissue.

In the first patient in whom I encountered an intramedullary tumor, I determined to test the correctness of this theory (Case 1). At the operation, the swollen cord was incised, and the incision watched for several minutes. Soon there was to be seen a bulging of a small bit of tumor mass through the incision, and while we watched it, the protrusion gradually increased in size while the incision in the cord gradually lengthened. The muscles, fascia and skin were then closed by suture, and the patient sent back to her bed. One week later, the wound was reopened, and the entire tumor was found lying outside of the cord, so that after the division of a few adhesions the tumor could be removed with practically no touching of the cord itself. The patient made a very good recovery.

This case proved the correctness of the theoretical considerations, and we applied the method to other cases of intramedullary tumors. We have called the method "the method of extrusion."

Based upon the anatomical grounds described in the early part of this paper, and upon the physical process above described, I desire to recommend the method of extrusion for the treatment and removal of intramedullary tumors of the spinal cord. If, after laminectomy and incision of the dura, the surgeon finds that he has to deal with an intramedullary growth, he should make a short incision about 1 cm. long, in the posterior median column a few millimeters outside of the posterior median fissure, at the spot where the growth seems to be nearest the surface of the cord. The incision should be deep enough to divide the pia and the substance of the column down to the tumor. The tumor will then begin to bulge through the incision. No matter how markedly the tumor will protrude, the surgeon must not attempt to remove the growth, for fear of grave injury to the cord. The operation must be concluded for the time being, the dura left wide open, the muscles, fascia and skin carefully closed, and the actual removal of the growth left to a second operation. After about one week, the wound is reopened, and the tumor, which will in all probability be found outside of the cord, can be removed by dividing the few adhesions which remain. When the tumor has been removed, the opening in the pia may be closed by a few very fine (split-silk) sutures, and the dura, muscles, fascia, and skin closed in the usual manner.

If, at the second operation, the tumor is found to be still closely connected with cord tissues, it is advisable to leave it *in situ* and not attempt its removal. In one instance, an intramedullary fibrolipoma, I made the error of attempting to remove a small part of the tumor which was still closely connected with the cord, and caused the symptoms of a transverse lesion, which had existed before the extrusion of the tumor, to reappear. In several later cases, having learned my lesson, I allowed the tumors to remain and left the dura wide open. These patients have improved so much that a later operation seemed unjustifiable. Cushing has had a similar experience.

In the case of intramedullary tumors which extend over a large number of segments, and which infiltrate the cord substance, a small incision into the cord made in the manner above described at the level of the

most marked symptoms may allow of the partial extrusion of the tumor, with amelioration of the symptoms.

Up to the present time I have applied the principles of intramedullary surgery above laid down to eleven cases of intramedullary diseases—eight cases of intramedullary tumor, two cases of hydromyelia and intramedullary cyst formation and one case of syringomyelia with marked glia formation. In all of the patients who could be followed for long periods of time, there was a very decided and in some of them a very marked improvement. As most of the cases have been reported elsewhere, I shall give only the synopses of the histories in this place.

CASE 1. Intramedullary glioma of the cervical cord; laminectomy, incision of cord, extrusion of tumor, removal; recovery. Result, very marked improvement with return of power in all extremities. Patient returned to her work (Figs. 7, 8, and 9).

CASE 2. Intramedullary glioma of cervical cord; laminectomy, incision of cord, extrusion of tumor, removal, death in four hours from respiratory paralysis.

CASE 3. Intramedullary sarcoma of cervical cord; laminectomy (Dr. Beer); extrusion, removal, recovery. Result, marked improvement; later, recurrence, operation, removal of recurrent tumor mass; no improvement.

CASE 4. Infiltrating tumor (glioma) of upper cervical cord and medulla oblongata; laminectomy and incision of cord; partial extrusion of tumor; death after two days from respiratory paralysis. Post-mortem showed that the tumor extended into the medulla and had grown through the posterior surface into the posterior cranial fossa (Fig. 10).

CASE 5. Hydromyelia, laminectomy, incision of cord, drainage of cord cavity into subdural space. Result, great improvement of complete paralysis of lower and partial paralysis of upper extremities, so that patient is up and about and has returned to his work (Fig. 5).

CASE 6. Intramedullary tumor of cervical cord; laminectomy, incision of cord, partial extrusion of diffuse tumor of cord; some improvement.

CASE 7. Syringomyelia; gliosis cervicalis; laminectomy, incision of cord in cervicodorsal region for decompressive purposes; marked improvement in power of upper extremities (Fig. 11).

CASE 8. Diffuse meningomyelitis with intramedullary cyst; laminectomy, incision of cord, drainage of intramedullary cyst; no improvement. Death three months later from exhaustion; post-mortem showed extensive lesions throughout dorsal and lumbar cord.

CASE 9. Intramedullary lipoma of cervical cord; laminectomy, incision of cord, partial extrusion of tumor. Great improvement so that quadriplegic patient has recovered almost complete power in the upper and much power in the lower extremities.

CASE 10. Intramedullary fibrolipoma of cervical cord; laminectomy, incision of cord, extrusion of tumor, great improvement in symptoms with disappearance of paralysis of upper and improvement in paralysis of lower extremities. Later, removal of tumor, which is not entirely extruded from the cord, followed by symptoms of transverse myelitis. Thereafter slow improvement.

CASE 11. Intramedullary fibrocyst of cervical cord; laminectomy, incision of cord, extrusion of cyst, removal at second operation, almost complete recovery, continuous improvement (Figs. 13 and 14).

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THE HEART IN FIBROID TUMORS OF THE UTERUS

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THE frequent association of cardiac lesions with fibromyomata of the uterus, noted both clinically and at autopsy, has led to the belief that a causal relation exists between them. Many authorities have gone so far as to assume that there is a specific form of heart disease associated with these tumors, which has been designated as the fibroid heart. A review of the literature shows it to be particularly rich in the subject under discussion and with few exceptions the authors maintain the view of causal relationship. While all do not speak of a fibroid heart as an entity, still there is a more or less unanimity of opinion that such an entity exists. The universality of this opinion is expressed by Barrows (1) in a very recent article, who says, "In all the literature at my command I can find but two dissenting opinions, that of Neu (2) and McGlinn (3)." Other exceptions, however, appear in the literature; notably, McDonald (4), Jaschke (5), Neu and Wolf (6), Kelly and Noble (7), Kelly and Cullen (8), Brickner (9), Rosthorn (10), and Winter (11).

The belief of causal relationship and of a fibroid heart is based principally on (*a*) The frequent association of organic and functional heart lesions with uterine myomata; (*b*) deaths both before and after operation, explainable only by heart failure; (*c*) the study of a comparatively few post-mortem records of such deaths; (*d*) conclusions based on various theories advanced to explain the causal relationship and the mode of production of the fibroid heart. The literature in support of this view is too voluminous to quote in detail, but for the convenience of the readers a partial bibliography is appended. The more important articles in this class are those by Shoemaker (12), Baldy (13), Williams (14), Doane (15), Barrows, Fleck (16), Doran (17), Cumston (18), Sebileau (19) and Boldt (20).

The heart lesions most frequently described as being found are brown atrophy, fatty degeneration of the myocardium, cloudy swelling, myocarditis, hypertrophy, dilatation, arrhythmia and valvular disease. It is not the purpose of this paper to prove that there is no connection between uterine fibroids and heart disease, but it is intended as an effort to clarify the subject and place the connection upon a rational basis. That changes take place in the heart as a direct result of uterine myomata is not to be denied. My belief in the matter is that there is no proof of the existence of a distinct fibroid heart and that all the changes which are commonly found can be explained by what might be termed accidents of the presence of the tumor. Further, while we believe that hæmorrhages, infections, degenerations, and resistance to circulation by pressure on pelvic vessels and by large tumors do produce cardiac changes, the general assumption that all the cases of degenerations, hypertrophy, dilatation, myocarditis and sclerotic changes found in connection with uterine myomata are due to the tumor not only lacks confirmation, but the weight of the evidence is against the acceptance of this view.

The present study is based on the records of 2700 autopsies made at the Philadelphia General Hospital and is supplemented by a study of 3000 autopsy records from the same source, previously reported in the New York Medical Journal of June 3, 1911. Of the 2700 records, 632 were those of female bodies, in 131 of which fibroid tumors of the uterus were found. In the paper previously reported, 113 tumors were studied, making a total of 244 cases upon which to base this report.

Table I represents the report of 131 tumors studied from the standpoints of age, race, clinical diagnosis, position of the tumor, size of the tumor, associated heart lesions and associated pathological lesions of other organs.

Table II shows the heart lesions found in connection with 100 cases of uterine myomata compared with the heart lesions found in 100 other cases of the same age and race in which such tumors were not present.

The subsequent tables show these 100 cases

of fibroid tumors of the uterus, divided into ten-year age periods, compared with equal numbers of non-fibroid cases of the same ages and race.

The method of collecting the cases without fibroids for comparison was as follows: When a case with a uterine fibroid was found in the records, the age and race were noted. The records were then searched consecutively until a report was found of a woman of the same age and race without a fibroid. No attempt was made to select cases, so that I believe these tables offer an impartial basis for comparison as to the frequency and character of heart lesions in women with and without fibroid tumors of the uterus.

In the final table, 13 additional cases between the ages of 20 and 40 are added to the 15 in the original series found between these ages. The purpose of this was to eliminate the age factor as far as possible in the causation of certain lesions. It was felt that 15 cases hardly presented a fair basis for comparison.

In the 632 female bodies uterine fibroids were found 131 times, or in 20.57 per cent, a rather remarkable showing of the frequency of these tumors. Of the 131 women, 74 were white, 56 negroes, and 1 Indian.

The average age of the women was 40.5 years, divided in age periods as follows:

20 to 29 years,	5 cases,	3.84 per cent
30 to 39 years,	17 cases,	13.08 per cent
40 to 49 years,	32 cases,	24.61 per cent
50 to 59 years,	30 cases,	23.07 per cent
60 to 69 years,	32 cases,	23.84 per cent
70 to 79 years,	9 cases,	6.94 per cent
80 years and over,	6 cases,	4.62 per cent

It is rather startling to note that out of the 131 cases but three (1100, 1938, 2527) were diagnosed clinically as fibroids of the uterus. A résumé of the cases, however, will show that with but few exceptions the tumors were so small or so situated that it would have been difficult to make a diagnosis even if a pelvic examination had been made. The relatively small size of the tumors, I think, gives added interest to this study, because if there are changes in the heart depending upon the presence of the tumors it shows that the size and situation of the neoplasms play little or no part in the production of the changes.

The heart lesions found in the cases in Table I are as follows:—

Normal hearts	7
Hypertrophy of left ventricle	4
Hypertrophy and dilatation of both ventricles	1
Dilatation of left ventricle	3
General hypertrophy	35
Hypertrophy and dilatation of left ventricle	2
General dilatation	25
Dilatation of left auricle	1
Dilatation of right auricle	2
Myocarditis	27
Brown induration	1
Soft myocardium	3
Cloudy swelling	10
Aortic sclerosis	37
Pulmonic sclerosis	1
Atheroma of aorta	19
General sclerotic changes	2
Chronic endocarditis	11
Acute pericarditis	1
Hydropericardium	3
Patulous foramen ovale	1
Syphilis of heart	1
Dilatation of right heart	1
Fatty degeneration	27
Brown atrophy	5
Pale myocardium	3
Mitral sclerosis	45
Tricuspid sclerosis	4
Chronic valvulitis	12
Coronaries sclerotic	11
Acute endocarditis	2
Subendocardial hæmorrhage	1
Chronic pericarditis	6
Tubercular pericarditis	1
Atrophy of heart	1
Sarcoma of heart	1
Total variety of lesions	35
Total number of lesions	300
	— 310

In considering the possible relation of the fibroid tumors to these lesions it may be well to discuss at the same time the various theories which have been advanced to explain the relationship. Among which are:—

First. Hæmorrhages become the cause of great weakness, and the heart, being poorly nourished, degenerates.

Second. Anæmia resulting from hæmorrhages is responsible for the degenerations and dilatations.

Third. More or less constant loss of blood will produce fatty degeneration and brown atrophy.

Fourth. Changes in the heart muscle may result from alterations in the blood pressure incident to hæmorrhages.

Fifth. A toxin produced during the growth of the tumor causes degenerative changes in the heart and blood-vessels.

Sixth. Fibroid tumors of the uterus are only a local feature of a general process.

Seventh. Poisonous products are produced by the grossly changed ovaries present in fibroid tumors, and these, acting on the heart, cause the various degenerations.

Eighth. Heart lesions are secondary to changes produced in the kidneys by pressure on the ureters.

Ninth. Hypertrophy results because the heart has added work to perform to supply the tumor with blood.

Tenth. Pressure on the vessels in the pelvis increases circulatory resistance and leads to hypertrophy of the heart.

Eleventh. Infections of the tumor may lead to secondary degenerative heart changes.

Twelfth. Infections of the bladder may ascend and affect the kidneys and in turn give rise to degenerative heart changes.

Thirteenth. Pain influences the regularity of the heart's action. Pain in fibroids produces arrhythmia.

Fourteenth. Pressure of the tumor on the sympathetics may produce arrhythmia.

Fifteenth. "A reflex from the uterus may influence by centripetal action a certain point of the nervous centers which is transmitted centrifugally to the arterioles; from this results a contraction of these arterioles, an exaggeration of pressure in the small circulation, and an excess of work to the heart to which it succumbs" (Potain).

In considering the possibility of the existence of a fibroid heart, all these theories can be dismissed with the exception of the fifth and sixth. Manifestly none of the others are applicable to all cases of fibroid tumors.

Considering, then, the only two rational theories, namely, that the tumor, in its growth develops a toxin which affects the heart, and that the fibroid tumor is only a local manifestation of a general process, it is reasonable to assume that if either of these theories were true, the heart would show some distinct change which would be common to all cases. This study proves that this is not so. We find in Table I that there are 35 distinct lesions, no one of which (mitral sclerosis) occurs in more than 45 of the cases. The same thing is true in the second series of 100 cases (Table II) in which there are 30 distinct lesions no one of which (mitral sclerosis) occurs in more than 57 of the cases.

Again, if the heart changes were dependent upon a toxin elaborated by the tumor, we should expect to find the extent of the changes depending upon the size of the tumor, as it is reasonable to assume that a large tumor would generate more toxic matter than a small one. *Per contra*, if a fibroid tumor of the uterus is simply a local manifestation of a general process, we should find the largest tumors in those cases which show the greatest changes in the cardiovascular system. A study of Table I does not bear out these contentions. Thus in case No. 525, with one subserous tumor measuring 1.5 cm. in diameter, the heart weighed 550 gms. and showed hypertrophy and fatty degeneration, while in No. 1100, with a tumor weighing 1630 gms., the heart weighed but 320 gms. and the only change recorded was myocarditis. Similar comparisons can be made between Nos. 260 and 511; 937 and 989; 1707 and 1951; 2160 and 2186, etc.

It may be argued that in both these series there is a more or less constant change in the cardiovascular system; namely, that of sclerosis. To a certain extent this is true; thus, in Tables I and II, totaling 231 cases, we find 541 lesions of which 326, or 60.3 per cent, are sclerotic in character. If we study the first series we find that of the 131 cases, 108, or 82.44 per cent, occurred after the age of 40 years, a period in which sclerotic changes of the cardiovascular system are the rule and not the exception. This relationship of age to sclerotic changes in the heart and vascular system is best shown in the second series under Tables III, IV, V, VI, VII, VIII, IX, X. These tables are arranged in various age groups of ten years to show the heart changes in fibroid tumors at various ages compared with the heart changes in an equal number of non-fibroid cases of the same age and race.

In the second series of 100 cases of fibroids 195 sclerotic lesions were found and exactly the same number were present in the non-fibroid cases. In all the age periods beyond 40 years, with the exception of the period between 70 and 79, not only were the sclerotic

changes more common in the non-fibroid cases, but all cardiac lesions were in excess of the fibroid ones.

In Table X, which represents 28 cases of fibroids compared with the same number of non-fibroids between the ages of 20 and 40 years, we find that the heart lesions in the fibroid cases are far in excess of those in the non-fibroid ones. From this it may be argued that eliminating the age factor, fibroids do produce sclerotic changes. This inference I do not believe to be justified. The number of cases for comparison is entirely too small upon which to base a just conclusion. Again, if it is argued that fibroids do produce sclerotic changes of the heart, they must be considered selected cases and if compared with another series of selected cases, such as interstitial nephritis, the heart lesions in the latter will far outnumber those found in the former. The number of non-fibroid cases in this table could not have been better selected to show the absence of sclerotic changes, as in only 6 of them (uræmia, 4; syphilis, 2) were any lesions present which could produce such changes.

In the two series there were 50 cases of fibroids between the ages of 20 and 40. Sclerotic changes were noted in 22 of these cases and absent in 28. In Table I, the largest tumors were in those cases which showed no sclerotic changes. The smallest tumors were associated with the most marked sclerotic changes. Again, in Table I, the sclerotic lesions numbered 17, divided among 11 cases. In these same cases were such lesions as syphilis, arteriosclerosis, cirrhosis of the liver, and acute and chronic nephritis which might be held responsible for the sclerotic changes in the heart. Therefore, in view of the fact that sclerotic changes in the heart were not a constant finding, that the largest tumors were not associated with such changes, and that other lesions were present to account for the scleroses, I believe that we are justified in assuming that no relationship between them can be proven.

No other one lesion occurs frequently enough to be considered as a typical heart change depending upon the presence of the uterine tumor. Let us consider briefly, however, those lesions which are most frequently ascribed to the fibroid; hypertrophy, dilatation, fatty degeneration, brown atrophy, cloudy swelling, and myocarditis.

Hypertrophy was found as follows in the first series of cases: General, 35; hypertrophy of left ventricle, 4; hypertrophy with dilatation of the left ventricle, 2; hypertrophy with dilatation of both ventricles, 1; a total of 42 times. Theories eight and nine can only be considered in the discussion of the cause of hypertrophy; theory fifteen is entirely too fanciful to receive serious consideration. In only six of the cases in Table I (512, 551, 1003, 1938, 1991, 2186) was the tumor of such a size as to increase the work of the heart, or so situated as to cause pressure and increase circulatory resistance. In none of the other cases was the tumor larger than 4 cm. and in the majority of cases it was 2 cm. or less. It is evident, then, that the hypertrophy in the other 36 cases must have been independent of the fibroid. A little study of Table I will show that in these cases the tumor has nothing to do with the production of cardiac hypertrophy and that it was caused by conditions entirely foreign to the myomata. Thus in case No. 509 we find the hypertrophy due to disease of the mitral valve and arteriosclerosis of the kidneys. Again in case No. 512, the lesion was secondary to mitral and aortic valvular disease and sclerotic kidneys. Practically the same causes can be shown in Nos. 551, 891, 1051, 1134, 1313, 1680, etc. In other cases different lesions can be shown to have been the cause. Thus in cases Nos. 709, 937 and 1011 atrophic cirrhosis of the liver was the underlying cause of the hypertrophy. In cases Nos. 1026, 1051, 1134, 1324, 1476, etc., the primary cause was arteriosclerosis. Other cases can be cited in Table I, where still other conditions, entirely foreign to the myomata, were the cause of the cardiac hypertrophy. Table II, shows that hypertrophy was practically as frequently met with in the non-fibroid as in the fibroid cases—29 in the former and 34 in the latter.

While we can accept the theory that fibroid tumors of the uterus do cause cardiac hypertrophy I believe that we must conclude that all cases of hypertrophy found in connection

with such tumors are not produced by them and that the only manner in which they can produce such a heart lesion is in the manner set forth in theories numbers eight and nine.

In Table I cardiac dilatation affecting the whole or a part of the heart was present in 32 of the cases. In Table II dilatation was noted in 9 of the fibroid cases and in 21 of the non-fibroid. Such an unstable lesion can hardly be considered as a specific form of cardiac disease, especially as it occurred more frequently in cases without tumors than in those with tumors. That it may be secondary to hypertrophy and fatty degeneration, which in some cases are dependent upon the tumor, is admitted, but that its presence can always be accounted for by the fibroid is denied. The usual cause of cardiac dilatation is valvular disease. In Table I dilated hearts are noted in cases Nos. 709, 1388, 1476, 1760, 1951, 2549, 2660, and 2856, which are independent of sclerotic valvular disease, and are apparently dependent upon fatty degeneration of the myocardium. In 5 of these cases (1388, 1476, 1760, 2660, 2856), however, the tumor was so small and so situated that it could hardly be responsible for the fatty degeneration. Thus in the entire series of cases we have but three examples of the lesion where the tumor was the likely cause.

Fatty degeneration of the heart was present in 27 of the 131 cases in the first series. This lesion is commonly found in connection with hypertrophy and dilatation, in anæmic and cachectic states, and after the age of 40. The mode of production of fatty degeneration of the heart by fibroid tumors of the uterus can only be explained by anæmia from loss of blood, infections of the tumor or surrounding structures, malignant degenerations, a specific toxin, and secondary to hypertrophy and dilatation. As we have already proven that hypertrophy and dilatation are seldom the direct result of the tumors, and that there is no reason to believe in the presence of a specific poison, and as no cases of malignant degeneration or infection are reported in this series, it remains but to show how many of the cases were dependent upon anæmia resulting from hæmorrhages. In ten of the 27 cases of fatty degeneration the tumor was so situated and of such a size as would likely produce hæmorrhage (Nos. 221, 571, 709, 713, 1488, 1528, 1850, 2220, 2773). In one of these cases (No. 1850) the anæmia was due to ulcerative enterocolitis, the case being recorded as one of pernicious anæmia due to the intestinal condition. In the other 9 cases the ages vary from 46 to 83 years, so that in some the hæmorrhages must have ceased and the body recovered from the anæmia. Ignoring these factors for the sake of argument, we have but 10 out of 131 cases in which the tumor could have been responsible in any way for the fatty degeneration. In Table II we find that this lesion was more common in the non-fibroid than in the fibroid cases.

A fair conclusion in reference to this lesion would be that while under certain circumstances fatty degeneration of the heart may be caused by fibroid tumors of the uterus, all cases of this lesion found in connection with such tumors cannot be ascribed to them.

Brown degeneration of the heart was noted in 6 cases in Table I, and in 7 in Table II; a total of 13 in 231 cases. The usual cause of this condition is old age and advanced valvular disease. In Table I, brown atrophy is found in cases Nos. 238, 811, 1254, 2349, 2557, and 2768. The ages of these cases are 65, 78, 57, 67, 84 and 53. In all except No. 238 advanced age or sclerotic valvular changes are present to account for the presence of the lesion. In case 238 the age (65) may be considered advanced enough in itself to account for the lesion but the fact that the woman also had advanced cancer and that the fibroid was very small would eliminate the tumor as the cause of the condition in this case. Thus in this entire series we do not find a single case of brown atrophy which was likely produced by the fibroid tumor. In fact, the lesion appears so seldom in connection with fibroid tumors that it can hardly be considered anything but a casual lesion.

Cloudy swelling of the myocardium is noted in ten of the cases in Table I, and in two in Table II, a total of 12 in the entire series. The usual cause of cloudy swelling is an intoxication. In all the cases in Table I (464, 1525, 1615, 1718, 1800, 2125, 2220, 2339, 2766, 2858) some disease capable of and usual-

ly producing severe systemic intoxication was present, peritonitis, pneumonia, tuberculosis, and nephritis. With these diseases present to produce the condition and it being so infrequent, I hardly believe that it can ever be ascribed to fibroids except in event of infections or malignant degenerations.

Chronic myocarditis occurs in 27 of the cases in Table I, and in 15 in Table II, a total of 42 times in 231 cases. In Table II it is shown that chronic myocarditis is no more common in fibroid than non-fibroid cases. Chronic or fibroid myocarditis is frequently associated with sclerotic changes in the coronary arteries and valves; it is common late in life and is secondary to various intoxications. While we may assume that myocarditis may result from infections and degenerations of a fibroid tumor, in order to prove that it did result entirely from the tumor in a given case we would have to eliminate the usual causes of its production. In Table I there is no record of infection of the tumor and no degeneration except calcification. In all the cases of myocarditis recorded there are lesions present in the heart or other organs which could produce the condition. We are safe in saying that in this series there is not a single case of myocarditis which could be attributable to the tumor. When we find that myocarditis is just as common in cases without fibroids as in those with them, and when infections and degenerations of the tumors which might produce it are so rare, it must be concluded that this lesion, depending entirely upon the presence of a myomata, must be extremely uncommon.

It is impossible from a study of this character to present much data in relation to the association of arrhythmia and with fibroids. That arrhythmia does occur is of course acknowledged, but is it not fair to assume that in the majority of cases it is a symptom of the myocarditis which is so frequently present and which bears no relation to the presence of the tumor?

Other heart lesions, not usually described, may result from a fibroid tumor, such as infections of the endocardium and pericardium from infections of the tumor, and metastases in the case of malignant degeneration, but these would be extremely rare and would be purely accidental.

The number of cases studied in this report is, I believe, sufficient upon which to base the following conclusions:

First. From this report a definite entity of a fibroid heart cannot be sustained.

Second. If the fibroid tumors of the uterus were the cause of all the heart lesions described in this study, then every tumor, regardless of its size and situation, should be removed—a contention that the most radical would hardly admit.

Third. Uterine myomata, occurring in middle and advanced life, are practically always associated with sclerotic heart lesions. These lesions are a part of a general process and bear no relation to the fibroid.

Fourth. Large tumors, by increasing the work of the heart, and tumors causing pressure on the pelvic circulation may produce hypertrophy and secondary dilatation of the heart.

Fifth. Anæmia, from hæmorrhages, infections and certain degenerations of the tumor, may affect the heart secondarily, causing changes such as fatty degeneration, brown atrophy, and cloudy swelling.

Sixth. The majority of cases of fatty degeneration, brown atrophy, cloudy swelling, myocarditis, etc., found in connection with fibroid tumors of the uterus are not caused by the tumor, but by conditions entirely foreign to the tumor.

TABLE I

Autopsy No. 221. White, age 53. Clinical diagnosis: Hemiplegia, uræmia. Position of tumor: One submucous polyp. Size of tumor: 1 cm. Heart condition: Weight (?), fatty. Associated pathological findings: General arteriosclerosis; chronic interstitial nephritis.

Autopsy No. 238. White, age 65. Clinical diagnosis: Cholecystitis. Position of tumor: One interstitial. Size of tumor: 2 cm. Heart condition: Weight, 145 gms. Brown induration of myocardium. Associated pathological findings: Cancer of kidney; metastases to liver, lung and pleura.

Autopsy No. 244. White, age 59. Clinical diagnosis: Cancer of stomach. Position of tumor: Few interstitial; one submucous. Size of tumor: Small. Heart condition: Weight, 320 gms. Interstitial myocarditis; mitral sclerosis. Associated

pathological findings: Cancer of sigmoid; metastases to liver; fatty liver; gall-stones; chronic parenchymatous nephritis.

Autopsy No. 260. White, age 66. Clinical diagnosis: Abdominal aneurism; arteriosclerosis. Position of tumor: One subperitoneal. Size of tumor: Small. Heart condition: Weight, 480 gms. Acute vegetative endocarditis; chronic endocarditis. Associated pathological findings: Arteriosclerosis.

Autopsy No. 262. White, age 42. Clinical diagnosis: Cirrhosis of liver. Position of tumor: One subserous. Size of tumor: 1¾ cm. Heart condition: Weight, 310 gms. Pericarditis; mitral sclerosis. Associated pathological findings: Gall-stones; cirrhosis of liver; chronic interstitial nephritis.

Autopsy No. 264. Black, age 30. Clinical diagnosis: Pulmonary tuberculosis. Position of tumor: One subserous. Size of tumor: 5 cm. Heart condition: Weight, 300 gms. Hydropericardium; sclerotic mitral and tricuspid valves; myocarditis. Associated pathological findings: Miliary tuberculosis; subacute nephritis; acute gastritis; tubercular enteritis.

Autopsy No. 345. White, age 78. Clinical diagnosis: Mitral regurgitation. Position of tumor: One subserous. Size of tumor: 3 cm. Heart condition: Weight, 380 gms. Myocarditis; chronic mitral endocarditis; atheroma of aorta; patulous foramen ovale. Associated pathological findings: Chronic diffuse nephritis.

Autopsy No. 464. Black, age 42. Clinical diagnosis: Tubercular peritonitis. Position of tumor: Multiple. Size of tumor: (?) Heart condition: Weight, (?); Cloudy swelling. Associated pathological findings: chronic purulent peritonitis.

Autopsy No. 478. White, age 46. Clinical diagnosis: Hemiplegia; myocarditis. Position of tumor: One subserous. Size of tumor: Small. Heart condition: Weight, 290 gms. Sclerotic mitral and aortic valves; atheroma of aorta. Associated pathological findings: Œdema of meninges.

Autopsy No. 509. Black, age 42. Clinical diagnosis: Nephritis; Œdema of lungs. Position of tumor: Many interstitial and subserous. Size of tumor: Small. Heart condition: Weight, 400 gms. Hydropericardium; hypertrophy and dilatation of left ventricle; sclerosis of mitral valve and coronary arteries. Associated pathological findings: Gall-stones; arteriosclerotic kidneys; tubercular mesenteric lymph nodes.

Autopsy No. 511. Black, age 45. Clinical diagnosis: Chronic nephritis. Position of tumor: One subserous. Size of tumor: Large. Heart condition: Weight, 300 gms. Normal heart. Associated pathological findings: Acute parenchymatous nephritis.

Autopsy No. 512. Black, age 56. Clinical diagnosis: Uræmia; endocarditis. Position of tumor: One interstitial. Size of tumor: Large. Heart condition: Weight, (?). Hypertrophy and dilatation of ventricles; sclerosis of mitral and aortic valves; atheroma of aorta. Associated pathological findings: Hæmorrhagic pancreatitis; arteriosclerotic kidneys.

Autopsy No. 520. Black, age 75. Clinical diagnosis: Pericarditis; pneumonia. Position of tumor: Two subserous. Size of tumor: Walnut. Heart condition: Weight, 350 gms. Hypertrophy; pericarditis; atheroma of aorta. Associated pathological findings: Echinococcus cyst of liver; arteriosclerosis of kidneys.

Autopsy No. 525. White, age 48. Clinical diagnosis: Myocarditis; nephritis; anasarca. Position of tumor: One subserous. Size of tumor: 1½ cm. Heart condition: Weight, 550 gms. Hypertrophy; fatty degeneration. Associated pathological findings: Gall-stones; fatty liver; obesity; chronic nephritis.

Autopsy No. 537. White, age 44. Clinical diagnosis; Transverse myelitis. Position of tumor: One subserous. Size of tumor: Small. Heart condition: Weight, 250 gms. Slight sclerosis of aortic valve. Associated pathological findings: Fracture of spine; arteriosclerosis.

Autopsy No. 551. White, age 66. Clinical diagnosis: Myocarditis; chronic interstitial nephritis. Position of tumor: multiple. Size of tumor: Largest, 10 cm. Heart condition: Weight, 850 gms. Hypertrophy; sclerosis of mitral and aortic valves; atheroma of aorta. Associated pathological findings: Nutmeg liver; chronic nephritis; hydrothorax.

Autopsy No. 571. White, age 49. Clinical diagnosis: Myocarditis; pneumonia; anæmia; chronic nephritis. Position of tumor: One submucous polyp. Size of tumor: 4 cm. Heart condition: Weight, 360 gms. Fatty infiltration. Associated pathological findings: Arteriosclerosis; chronic interstitial nephritis; fatty liver; gastritis; acute enteritis.

Autopsy No. 589. Black, age 34. Clinical diagnosis: Cerebrospinal syphilis; phthisis; nephritis. Position of tumor: One subserous. Size of tumor: Large. Heart condition: Weight, 230 gms. Soft and flabby myocardium. Associated pathological findings: Cancer of lung, liver, and pancreas; chronic diffuse nephritis.

Autopsy No. 616. White, age 64. Clinical diagnosis: Pneumonia; myocarditis; nephritis. Position of tumor: One interstitial. Size of tumor: 1½ cm. Heart condition: Weight, 320 gms. Chronic myocarditis; fatty degeneration. Associated pathological findings: Chronic interstitial nephritis with acute exacerbation; fatty liver.

Autopsy No. 666. Black, age 34. Clinical diagnosis: Myocarditis; nephritis. Position of tumor: One subserous polyp. Size of tumor: ½ in. Heart condition: Weight, 200 gms. Myocarditis; valvular endocarditis; atheroma of aorta. Associated pathological findings: Atrophic cirrhosis of liver; chronic interstitial nephritis; enteritis.

Autopsy No. 685. Black, age 53. Clinical diagnosis: Myocarditis; chronic nephritis. Posi-

tion of tumor: Two submucous polyps. Size of tumor: 1 cm. Heart condition: Weight, 530 gms. Dilatation; sclerotic changes. Associated pathological findings: Arteriosclerosis; diffuse nephritis; papilloma of ovary.

Autopsy No. 688. Black, age 38. Clinical diagnosis: Myocarditis; nephritis. Position of tumor: One submucous. Size of tumor: 4 cm. Heart condition: Weight, 520 gms. Fibroid myocarditis; hypertrophy of left ventricle. Associated pathological findings: Pleurisy with effusion; chronic diffuse nephritis.

Autopsy No. 709. White, age 83. Clinical diagnosis: Jacksonian epilepsy; endocarditis; nephritis. Position of tumor: Several submucous polyps. Size of tumor: Small. Heart condition: Weight, 500 gms. Fatty heart; hypertrophy and dilatation. Associated pathological findings: Chronic interstitial nephritis; cirrhosis of liver; softening of brain.

Autopsy No. 713. White, age 75. Clinical diagnosis: Myocarditis; nephritis; arteriosclerosis. Position of tumor: One submucous. Size of tumor: 1½ x 2 cm. Heart condition. Weight, 250 gms. Fatty degeneration. Associated pathological findings: Chronic interstitial nephritis.

Autopsy No. 764. White, age 55. Clinical diagnosis: Nephritis; endocarditis; arteriosclerosis; pneumonia; pseudobulbar palsy. Position of tumor: One submucous. Size of tumor: 1½ cm. Heart condition: Weight, 320 gms. Fatty infiltration. Associated pathological findings: Chronic diffuse nephritis; arteriosclerosis.

Autopsy No. 811. White, age 78. Clinical diagnosis: Œdema of lungs; arteriosclerosis. Position of tumor: One submucous polyp. Size of tumor: 4 cm. Heart condition: Weight, 280 gms. Brown atrophy; chronic endocarditis. Associated pathological findings: General arteriosclerosis; chronic interstitial and parenchymatous nephritis.

Autopsy No. 852. Black, age 69. Clinical diagnosis, Hemiplegia; chronic interstitial nephritis; mitral valvulitis. Position of tumor: Multiple. Size of tumor: (?). Heart condition: Weight, 290 gms. Chronic endocarditis; aortic sclerosis. Associated pathological findings: Parenchymatous nephritis.

Autopsy No. 867. White, age 43. Clinical diagnosis: Alcoholism; delirium tremens. Position of tumor: One interstitial. Size of tumor: 2 cm. Heart condition: Weight, 260 gms. Fatty heart; chronic valvulitis. Associated pathological findings: Fatty kidneys; colitis; gastritis.

Autopsy No. 891. White, age 29. Clinical diagnosis: Myocarditis; hypertrophy. Position of tumor: Two subperitoneal. Size of tumor: Millet seed. Heart condition: Weight, 360 gms. Mitral stenosis and regurgitation; hypertrophy and dilatation. Associated pathological findings: Subacute hæmorrhagic nephritis. Passive congestion kidneys.

Autopsy No. 903. White, age 61. Clinical diagnosis: Brain tumor. Position of tumor: Four subperitoneal. Size of tumor: Walnut. Heart condition: Weight, 260 gms. Chronic valvulitis. Associated pathological findings: chronic interstitial nephritis.

Autopsy No. 937. Black, age 41. Clinical diagnosis: Myocarditis; cirrhosis of liver; chronic parenchymatous nephritis. Position of tumor: Numerous. Size of tumor: Small. Heart condition: Weight, 520 gms. Hypertrophy; hydropericardium; endocarditis; myocarditis; atheroma of aorta. Associated pathological findings: Syphilitic cirrhosis of liver; ascites; passive congestion of kidneys.

Autopsy No. 989. Black, age 59. Clinical diagnosis: Fibroid of uterus; T. B. of lungs; chronic nephritis; tricuspid regurgitation. Position of tumor: Several interstitial. Size of tumor: 15 x 10 cm. Heart condition: Weight, 380 gms. Chronic myocarditis; chronic endocarditis. Associated pathological findings: Chronic diffuse nephritis.

Autopsy No. 996. White, age 53. Clinical diagnosis: Cancer of stomach. Position of tumor: Several interstitial. Size of tumor: 1 cm. Heart condition: Weight, 240 gms. Moderate fatty degeneration; mitral and aortic valvulitis; atheroma of aorta. Associated pathological findings: Chronic diffuse nephritis; gall-stones.

Autopsy No. 1003. White, age 46. Clinical diagnosis: Nephritis; aneurism of innominate. Position of tumor: One subperitoneal. Size of tumor: Large. Heart condition: Weight, 480 gms. Hypertrophy and dilatation; chronic aortic valvulitis; chronic pericarditis.

Autopsy No. 1010. White, age 47. Clinical diagnosis: Myocarditis; cancer of omentum. Position of tumor: Multiple. Size of tumor: Largest, 3 cm. Heart condition: Weight, 270 gms. Early myocarditis. Associated pathological findings: Cancer of ovary and omentum; chronic diffuse nephritis.

Autopsy No. 1011. Black, age 36. Clinical diagnosis: Myocarditis; hypertrophy; mitral and aortic valvulitis. Position of tumor: Many subserous; one interstitial. Size of tumor: Largest, 4 cm. Heart condition: Weight, 610 gms. Hypertrophy of left ventricle; incompetent mitral and aortic valves. Associated pathological findings: Cirrhosis of liver; chronic diffuse nephritis.

Autopsy No. 1018. White, age 61. Clinical diagnosis: Senility; arteriosclerosis. Position of tumor: One subserous calcified. Size of tumor: Small. Heart condition: Weight, 240 gms. Fatty heart; chronic aortic valvulitis. Associated pathological findings: Pneumonia; tuberculosis of lungs and kidneys; chronic diffuse nephritis.

Autopsy No. 1026. White, age 65. Clinical diagnosis: Hemiplegia; myocarditis; nephritis; arteriosclerosis. Position of tumor: Multiple. Size of tumor: Largest, 5 x 3 cm. Heart condition: Weight, 410 gms. Hypertrophy left ventricle. Associated pathological findings: General arteriosclerosis; pneumonia; chronic diffuse nephritis; cerebral hæmorrhage.

Autopsy No. 1051. Black, age 61. Clinical diagnosis: Hemiplegia; myocarditis; arteriosclero-

sis. Position of tumor: One intraligamentous. Size of tumor: Small. Heart condition: Weight, 520 gms. Hypertrophy; chronic valvulitis; tuberculous pericarditis. Associated pathological findings: General arteriosclerosis; subacute diffuse nephritis; tubercular mediastinal lymph nodes.

Autopsy No. 1056. White, age 66. Clinical diagnosis: Dysentery; enteritis; myocarditis; arteriosclerosis. Position of tumor: One submucous polyp. Size of tumor: Small. Heart condition: Weight, 270 gms. Chronic valvulitis. Associated pathological findings: Ulcerative enteritis.

Autopsy No. 1081. Black, age 34. Clinical diagnosis: Tuberculosis of lungs and peritoneum. Position of tumor: One subperitoneal. Size of tumor: 2 cm. Heart condition: Weight, 310 gms. No lesions. Associated pathological findings: Chronic parenchymatous nephritis; fatty liver.

Autopsy No. 1098. White, age 54. Clinical diagnosis: Tuberculosis of lungs. Position of tumor: One. Size of tumor: $\frac{3}{16}$ in. Heart condition: Weight, 290 gms. No lesions. Associated pathological findings: Tuberculosis of lungs.

Autopsy No. 1100. Black, age 30. Clinical diagnosis: Fibroid of uterus. Position of tumor: One subserous. Size of tumor: Weight, 1630 gms. Heart condition: Weight, 320 gms. Myocarditis. Associated pathological findings: Parenchymatous nephritis.

Autopsy No. 1134. Black, age 35. Clinical diagnosis: Uræmia; parenchymatous nephritis. Position of tumor: Four subperitoneal; two submucous. Size of tumor: 1½ to 2½ cm. Heart condition: Weight, 400 gms. Hypertrophy; chronic valvular disease. Associated pathological findings: Chronic diffuse nephritis; arteriosclerosis; cerebral hæmorrhage.

Autopsy No. 1243. Black, age 42. Clinical diagnosis: Cerebral tumor; nephritis; myocarditis. Position of tumor: Two subperitoneal. Size of tumor: 2 cm. Heart condition: Weight, 290 gms. Chronic myocarditis; chronic valvulitis; sclerosis of coronaries. Associated pathological findings: Arteriosclerosis; chronic interstitial nephritis.

Autopsy No. 1252. Black, age 65. Clinical diagnosis: Hemiplegia; pneumonia; endocarditis; myocarditis; nephritis. Position of tumor: Many subperitoneal. Size of tumor: Large and small. Heart condition: Weight, 320 gms. Sclerotic aortic valve; atheroma of aorta. Associated pathological findings: Chronic interstitial nephritis.

Autopsy No. 1254. Indian, age 57. Clinical diagnosis: Pneumonia; chronic nephritis. Position of tumor: One. Size of tumor: Small. Heart condition: Weight, 280 gms. Muscle brown and flabby; slight sclerosis of mitral valve. Associated pathological findings: Pneumonia; gall-stones; diffuse nephritis.

Autopsy No. 1313. White, age 60. Clinical diagnosis: Arteriosclerosis; uræmia. Position of tumor: Two subperitoneal. Size of tumor: Chestnut. Heart condition: Weight, 520 gms. Fatty; dilatation and hypertrophy; sclerosis of aortic valve. Associated pathological findings: Tumors of right kidney, sternum, left lung; chronic parenchymatous nephritis.

Autopsy No. 1320. White, age 104. Clinical diagnosis: Myocarditis; nephritis; senility. Position of tumor: One. Size of tumor: Egg. Heart condition: Remarkably normal heart for age. Associated pathological findings: Pneumonia; stone in kidney; mystic kidney.

Autopsy No. 1324. Black, age 42. Clinical diagnosis: Mitral and aortic disease. Position of tumor: One subperitoneal. Size of tumor: Small. Heart condition: Weight, 600 gms. Hypertrophy and dilatation. Associated pathological findings: General arteriosclerosis; chronic interstitial nephritis.

Autopsy No. 1350. Black, age 40. Clinical diagnosis: Pulmonary and meningeal tuberculosis. Position of tumor: Multiple. Size of tumor: (?). Heart condition: Weight, 230 gms. Slight pulmonic sclerosis. Associated pathological findings: Miliary tuberculosis.

Autopsy No. 1388. Black, age 50. Clinical diagnosis: Acute dilatation of heart; laryngeal growth. Position of tumor: Multiple. Size of tumor: Large. Heart condition: Weight, 340 gms. Acute dilatation; myocarditis. Associated pathological findings: Gumma of lungs and vocal cords; passive congestion of kidneys.

Autopsy No. 1430. White, age 74. Clinical diagnosis: Myocarditis; arteriosclerosis; diffuse nephritis. Position of tumor: One subperitoneal nodule. Size of tumor: 1 cm. Heart condition: Weight, 530 gms. Chronic myocarditis; chronic indurated valvulitis. Associated pathological findings: General arteriosclerosis.

Autopsy No. 1476. White, age 65. Clinical diagnosis: Cerebral hæmorrhage; myocarditis; chronic diffuse nephritis; cardiac dilatation. Position of tumor: One interstitial nodule. Size of tumor: Pigeon egg. Heart condition: Weight (?). Hypertrophy and dilatation; fatty degeneration of heart. Associated pathological findings: Chronic diffuse nephritis; gall-stones; general arteriosclerosis; œdema of the brain.

Autopsy No. 1488. White, age 55. Clinical diagnosis: Pneumonia; uræmia. Position of tumor: Two interstitial. Size of tumor: 3 and 7 cm. Heart condition: Weight, 300 gms. Fatty degeneration. Associated pathological findings: Chronic diffuse nephritis; parenchymatous degeneration of liver; acute pancreatitis.

Autopsy No. 1504. White, age 60. Clinical diagnosis: Myocarditis; arteriosclerosis; chronic valvulitis; chronic diffuse nephritis. Position of tumor: Two subperitoneal. Size of tumor: 2 and 4 cm. Heart condition: Weight, 350 gms. Fatty degeneration; sclerosis of mitral and aortic valves; atheroma of aorta.

Autopsy No. 1525. Black, age 49. Clinical diagnosis: Apoplexy; myocarditis; pneumonia.

Position of tumor: Multiple; some necrotic. Size of tumor: 3 to 9 cm. Heart condition: Weight, 556 gms. Cloudy swelling. Associated pathological findings: Chronic diffuse nephritis; gall-stones; head not opened.

Autopsy No. 1526. White, age 81. Clinical diagnosis: Myocarditis; bronchitis; chronic interstitial nephritis. Position of tumor: Several submucous nodes. Size of tumor: 4 cm. Heart condition: Weight, 320 gms. Chronic myocarditis. Associated pathological findings: Chronic interstitial nephritis; fibroma of left kidney; hæmorrhage; cystitis.

Autopsy No. 1528. White, age 72. Clinical diagnosis: Cancer of uterus; myocarditis; diffuse nephritis; arteriosclerosis. Position of tumor: One large subperitoneal node. Many small submucous nodes which are gangrenous. Size of tumor: 2 to 6 cm. Heart condition: Weight, 300 gms. Fatty degeneration; atheroma of aorta.

Autopsy No. 1615. White, age 47. Clinical diagnosis: Pneumonia; uræmia. Position of tumor: One small submucous nodule. Size of tumor: Small. Heart condition: Weight, 250 gms. Cloudy swelling of myocardium. Associated pathological findings: Acute pneumonia; chronic interstitial nephritis.

Autopsy No. 1680. Black, age 50. Clinical diagnosis: Hypertrophy and dilatation of heart; myocarditis; chronic valvular disease; anasarca. Position of tumor: Three subperitoneal nodules. Size of tumor: 1½ cm. Heart condition: Weight, 840 gms. Valvular disease; hypertrophy. Associated pathological findings: Chronic nephritis; acute hæmorrhagic pancreatitis.

Autopsy No. 1707. White, age 86. Clinical diagnosis: Hemiplegia; aortic disease; arteriosclerosis. Position of tumor: Two subserous. Size of tumor: 1½ and ½ cm. Heart condition: Weight, 600 gms. Dilatation; sclerosis of aortic mitral and tricuspid valves. Associated pathological findings: General arteriosclerosis.

Autopsy No. 1718. White, age 41. Clinical diagnosis: General peritonitis; pneumonia. Position of tumor: One subserous. Size of tumor: ½ in. Heart condition: Weight, 320 gms. Cloudy swelling. Associated pathological findings: Acute pelvic peritonitis; acute parenchymatous nephritis.

Autopsy No. 1744. Black, age 45. Clinical diagnosis: Died two hours after admission; coroner's case. Position of tumor: One submucous nodule. Size of tumor: Marble. Heart condition: Weight, 580 gms. Hypertrophy and dilatation. Associated pathological findings: Acute nephritis: chronic diffuse nephritis; arteriosclerosis; cerebral hæmorrhage.

Autopsy No. 1759. Black, age 38. Clinical diagnosis: Endocarditis valvulitis; chronic interstitial nephritis. Position of tumor: Four subperitoneal nodules. Size of tumor: Small. Heart condition: Weight, 480 gms. Mitral valvulitis; hypertrophy and dilatation. Associated pathological findings: General arteriosclerosis; chronic interstitial nephritis; cancer of gall-bladder.

Autopsy No. 1760. White, age 43. Clinical diagnosis: Cerebral hæmorrhage. Position of tumor: One subperitoneal nodule. Size of tumor: 2 cm. Heart condition: Weight, 380 gms. Hypertrophy and dilatation; fatty degeneration. Associated pathological findings: Traumatic cerebral hæmorrhage.

Autopsy No. 1800. Black, age 38. Clinical diagnosis: Malignant tumor of bladder; tuberculosis of lungs. Position of tumor: One submucous. Size of tumor: Small. Heart condition: Weight, 300 gms. Cloudy swelling of myocardium. Associated pathological findings: Œdema of lungs.

Autopsy No. 1830. Black, age 69. Clinical diagnosis: Mitral and aortic disease; tuberculosis of lungs. Position of tumor: Interstitial. Size of tumor: Small. Heart condition: Weight, 310 gms. Mitral and aortic incompetence; tricuspid stenosis; chronic myocarditis. Associated pathological findings: Chronic diffuse nephritis; labor pneumonia.

Autopsy No. 1850. White, age 50. Clinical diagnosis: Progressive pernicious anæmia. Position of tumor: One submucous. Size of tumor: 1½ cm. Heart condition: Weight, 250 gms. Mitral and aortic sclerosis; fatty degeneration. Associated pathological findings: Atrophy of intestinal mucosa; healed ulcer of colon; fatty liver; fatty degeneration of kidneys.

Autopsy No. 1899. Black, age 80. Clinical diagnosis: (?). Position of tumor: One interstitial. Size of tumor: 8 x 6 cm. Heart condition: Weight, 250 gms. Fibroid myocarditis; fatty degeneration. Associated pathological findings: Chronic interstitial nephritis; hypernephroma right kidney; acute miliary tuberculosis.

Autopsy No. 1919. White, age 55. Clinical diagnosis: Uræmia. Position of tumor: One interstitial calcified. Size of tumor: 2½ cm. Heart condition: Weight, 500 gms. Fatty degeneration; atheroma of aorta. Associated pathological findings: Chronic interstitial nephritis with superimposed acute process.

Autopsy No. 1921. Black, age 28. Clinical diagnosis: Valvular heart disease. Position of tumor: Two interstitial. Size of tumor: 7 cm. Heart condition: Weight, 300 gms. Early fibroid myocarditis; atheroma of aorta. Associated pathological findings: Chronic interstitial nephritis; abscess of liver.

Autopsy No. 1937. White, age 61. Clinical diagnosis: Atrophic cirrhosis of liver; arteriosclerosis; tuberculosis of lungs. Position of tumor: Several interstitial. Size of tumor: Small. Heart condition: Weight, 260 gms. Fibroid myocarditis; fatty degeneration. Associated pathological findings: Atrophic cirrhosis of liver; chronic interstitial nephritis; emphysema of lungs.

Autopsy No. 1938. Black, age 59. Clinical diagnosis: Fibroid of uterus. Position of tumor: Large multiple. Size of tumor: 6 to 11 cm. Heart

condition: Weight, 370 gms. Hypertrophy; aortic and mitral stenosis. Associated pathological findings: Chronic interstitial nephritis with superimposed acute process; pneumonia; not operated on because of kidney condition.

Autopsy No. 1951. White, age 40. Clinical diagnosis: Coroner's case. Position of tumor: Numerous subserous and interstitial. Size of tumor: Largest 4 cm. Heart condition: Weight, 250 gms. Fatty degeneration; dilated left ventricle. Associated pathological findings: Glioma of brain.

Autopsy No. 1966. White, age 44. Clinical diagnosis: Cancer of stomach; mitral disease; hypertrophy; tuberculosis of lungs. Position of tumor: One submucous. Size of tumor: 1 cm. Heart condition: Weight, 220 gms. Mitral and aortic valvulitis. Associated pathological findings: Chronic interstitial nephritis; cancer of stomach.

Autopsy No. 1979. White, age 42. Clinical diagnosis: Myocarditis; acute nephritis. Position of tumor: One subserous; sarcomatous degeneration. Size of tumor: 2 cm. Heart condition: Weight, (?). Dilatation; mitral sclerosis. Associated pathological findings: Acute parenchymatous nephritis; anasarca.

Autopsy No. 1980. White, age 31. Clinical diagnosis: Dilatation of heart, acute; encephalitis; chronic nephritis. Position of tumor: One subserous. Size of tumor: Almond. Heart condition: Weight, 430 gms. Hypertrophy. Associated pathological findings: Chronic interstitial nephritis; early arteriosclerosis.

Autopsy No. 1991. Black, age 45. Clinical diagnosis: Cardiac dilatation and hypertrophy; anasarca. Position of tumor: Two subserous. Size of tumor: 15 cm. Heart condition: Weight, 550 gms. Dilatation and hypertrophy; sclerosis of mitral valve. Associated pathological findings: Chronic parenchymatous nephritis.

Autopsy No. 2022. White, age 63. Clinical diagnosis: Hemiplegia. Position of tumor: Two submucous. Size of tumor: 4 cm. Heart condition: Weight, 320 gms. Chronic interstitial myocarditis. Associated pathological findings: chronic interstitial nephritis; arteriosclerosis.

Autopsy No. 2058. White, age 68. Clinical diagnosis: Tabes dorsalis; chronic interstitial nephritis. Position of tumor: One subperitoneal. Size of tumor: (?). Heart condition: Weight, 260 gms. Chronic interstitial myocarditis; chronic endocarditis; sclerosis of mitral and aortic valves. Associated pathological findings: Chronic interstitial nephritis; general sclerotic changes in all organs.

Autopsy No. 2118. Black, age 30. Clinical diagnosis: Chronic myocarditis; arteriosclerosis; functional murmur; tuberculosis of lungs; chronic diffuse nephritis. Position of tumor: One subperitoneal. Size of tumor: 7½ x 5 cm. Heart condition: Weight, 250 gms. Muscle flabby, normal otherwise. Associated pathological findings: Chronic parenchymatous nephritis; congestion of lungs.

Autopsy No. 2125. Black, age 21. Clinical diagnosis: Puerperal sepsis. Position of tumor: Few subperitoneal. Size of tumor: Pea to marble. Heart condition: Weight, 260 gms. Cloudy swelling of myocardium; subendocardial hæmorrhage. Associated pathological findings: Pelvic peritonitis.

Autopsy No. 2132. Black, age 51. Clinical diagnosis: Ovarian cyst; ascites; myocarditis. Position of tumor: Four subperitoneal. Size of tumor: Largest 3 cm. Heart condition: Weight, 600 gms. Hypertrophy; dilatation; chronic mitral and tricuspid valvulitis. Associated pathological findings: Cirrhosis of liver; ovarian cyst; acute pyosalpinx; passive congestion of kidneys.

Autopsy No. 2160. Black, age 65. Clinical diagnosis: Chronic nephritis; mitral and aortic disease. Position of tumor: One interstitial. Size of tumor: ½ cm. Heart condition: Weight, 470 gms. Hypertrophy and dilatation; chronic mitral and aortic disease; chronic endocarditis. Associated pathological findings: chronic diffuse nephritis.

Autopsy No. 2183. White, age 55. Clinical diagnosis: Aortic disease; arteriosclerosis; tubercular peritonitis. Position of tumor: Multiple small nodules; calcareous. Size of tumor: Largest 3½ cm. Heart condition: Weight, 480 gms. Hypertrophy of left ventricle; dilated left auricle; chronic mitral valvulitis. Associated pathological findings: General arteriosclerosis; tubercular peritonitis.

Autopsy No. 2186. Black, age 46. Clinical diagnosis: Uræmia; mitral disease; chronic diffuse nephritis. Position of tumor: Cystic fibroid. Size of tumor: Weight, 1215 gms. Heart condition: 425 gms. Hypertrophy; calcified mitral valve atheroma of aorta. Associated pathological findings: Chronic interstitial and parenchymatous nephritis; cerebral hæmorrhage.

Autopsy No. 2220. White, age 44. Clinical diagnosis: Pneumonia. Position of tumor: Multiple. Size of tumor: Largest 4 cm. Heart condition: Weight, (?). Fat infiltration and cloudy swelling of myocardium; sclerosis of mitral and aortic valves. Associated pathological findings: General arteriosclerosis.

Autopsy No. 2258. Black, age 38. Clinical diagnosis: Anasarca; nephritis; œdema of lungs. Position of tumor: Two subserous nodules. Size of tumor: Small. Heart condition: Weight, (?). Hypertrophy and dilatation; chronic endocarditis. Associated pathological findings: Chronic interstitial nephritis.

Autopsy No. 2326. White, age 61. Clinical diagnosis: Sarcoma of liver. Position of tumor: Subperitoneal. Size of tumor: 5 cm. Heart condition: Weight, 260 gms. Muscle pale and soft; chronic mitral valvulitis. Associated pathological findings: Primary cancer of gall-bladder; gall-stones; small white kidneys.

Autopsy No. 2334. White, age 66. Clinical diagnosis: Hemiplegia; pneumonia; myocarditis. Position of tumor: Many small interstitial. Size of tumor: Small. Heart condition: Weight, 235

gms. Acute pericarditis; sclerosis of mitral and aortic valves coronary vessels. Associated pathological findings: Chronic interstitial nephritis; cranium not opened.

Autopsy No. 2346. White, age 60. Clinical diagnosis: Hemiplegia; myocarditis; chronic valvulitis. Position of tumor: One subperitoneal. Size of tumor: 1 cm. Heart condition: Weight, 565 gms. Hypertrophy and dilatation; sclerosis of mitral valve. Associated pathological findings: Chronic interstitial nephritis.

Autopsy No. 2349. White, age 67. Clinical diagnosis: Hemiplegia; valvulitis; dilatation; pneumonia. Position of tumor: One subperitoneal polyp. Size of tumor: ½ in. Heart condition: Weight, 415 gms. Hypertrophy; brown atrophy. Associated pathological findings: Chronic interstitial nephritis.

Autopsy No. 2350. White, age 67. Clinical diagnosis: Paralysis agitans. Position of tumor: Many subperitoneal nodules. Size of tumor: Small. Heart condition: Weight, 325 gms. Muscle normal; atheroma of all valves. Associated pathological findings: General arteriosclerosis.

Autopsy No. 2374. White, age 56. Clinical diagnosis: Diplegia. Position of tumor: Many subperitoneal nodules. Size of tumor: Pea to walnut. Heart condition: Weight, 300 gms. Normal.

Autopsy No. 2390. White, age 78. Clinical diagnosis: Pneumonia; arthritis deformans. Position of tumor: One submucous calcified. Size of tumor: 4 cm. Heart condition: Weight, 400 gms. Sclerosis of valves.

Autopsy No. 2394. Black, age 29. Clinical diagnosis: Mitral and aortic disease; nephritis. Position of tumor: Two interstitial. Size of tumor: Pea. Heart condition: Weight, 500 gms. Hypertrophy and dilatation; fatty degeneration of muscle; chronic mitral and aortic valvulitis. Associated pathological findings: Cyanotic induration of kidneys.

Autopsy No. 2399. White, age 63. Clinical diagnosis: Pneumonia. Position of tumor: Several subperitoneal. Size of tumor: 1 to 4 cm. Heart condition: Weight, 265 gms. Cloudy swelling of myocardium; sclerosis of mitral and aortic valves. Associated pathological findings: Chronic interstitial nephritis.

Autopsy No. 2401. White, age 65. Clinical diagnosis: Cerebral thromboses. Position of tumor: Several interstitial calcified. Size of tumor: Pea to marble. Heart condition: Weight, 320 gms. Fatty degeneration; sclerosis of mitral and aortic valves. Associated pathological findings: Syphilitic arteriosclerosis.

Autopsy No. 2418. Black, age 42. Clinical diagnosis: Paresis. Position of tumor: One subperitoneal. Size of tumor: 2 cm. Heart condition: Weight, 260 gms. Normal.

Autopsy No. 2494. Black, age 50. Clinical diagnosis: Myocarditis. Position of tumor: Two subperitoneal. Size of tumor: 1 cm. Heart condition: Weight, 300 gms. Fatty with moderate fibrosis; dilatation of left ventricle; chronic mitral and aortic valvulitis. Associated pathological findings: Chronic interstitial nephritis.

Autopsy No. 2527. Black, age 45. Clinical diagnosis: Uterine fibroid; acute dilatation of heart; diffuse nephritis; sent to hospital as case of eclampsia. Position of tumor: Multiple tumors. Size of tumor: Large. Heart condition: Weight, 510 gms. Chronic interstitial myocarditis; dilatation left ventricle; chronic aortitis. Associated pathological findings: Chronic interstitial nephritis; œdema of both lungs. Died day of admission to hospital of uræmia. No operation.

Autopsy No. 2549. Black, age 26. Clinical diagnosis: Parenchymatous nephritis; myocarditis; acute dilatation; anasarca. Position of tumor: One submucous. Size of tumor: 3 cm. Heart condition: Weight, 440 gms. Dilated right auricle. Associated pathological findings: Atrophic cirrhosis of liver; chronic interstitial nephritis; general œdema.

Autopsy No. 2557. White, age 84. Clinical diagnosis: Cancer of stomach. Position of tumor: Four interstitial; uterus atrophied. Size of tumor: 2½ cm. Heart condition: Weight, 275 gms. Brown atrophy; general sclerotic changes. Associated pathological findings: General arteriosclerosis; no cancer.

Autopsy No. 2581. White, age 65. Clinical diagnosis: Acute myocarditis. Position of tumor: One interstitial nodule. Size of tumor: Small. Heart condition: Weight, 745 gms. Hypertrophy and dilatation. Associated pathological findings: Chronic interstitial nephritis; atrophic cirrhosis of liver; gall-stones.

Autopsy No. 2590. Black, age 40. Clinical diagnosis: Myocarditis; aortic disease; diffuse nephritis. Position of tumor: Multiple. Size of tumor: (?). Heart condition: Weight, 475 gms. Acute endocarditis superimposed on chronic process; atheroma of aorta. Associated pathological findings: Diffuse nephritis.

Autopsy No. 2591. Black, age 63. Clinical diagnosis: Hemiplegia; uræmia; diffuse nephritis; myocarditis; arteriosclerosis. Position of tumor: Several interstitial calcified. Size of tumor: 3 cm. Heart condition: Weight, 455 gms. Chronic adhesive pericarditis; hypertrophy and dilatation; mitral stenosis; aortic endocarditis. Associated pathological findings: Chronic diffuse nephritis; gall-stones.

Autopsy No. 2594. White, age 32. Clinical diagnosis: General carcinomatosis. Position of tumor: One subperitoneal. Size of tumor: Small. Heart condition: Weight, 185 gms. Smaller than normal; sclerosis of coronaries; chronic pericarditis; sclerotic mitral and aortic valves. Associated pathological findings: General carcinomatosis; chronic interstitial nephritis.

Autopsy No. 2597. Black, age 55. Clinical diagnosis: Cardiac hypertrophy with dilatation. Position of tumor: One interstitial. Size of tumor:

2 cm. Heart condition: Weight, (?) gms. Hypertrophy; right auricle dilated; sclerosis of aortic and mitral valves. Associated pathological findings: Atrophic cirrhosis of liver; chronic interstitial nephritis.

Autopsy No. 2619. White, age (?). Clinical diagnosis: Uræmia; hypertrophy of heart. Position of tumor: One interstitial. Size of tumor: 2 cm. Heart condition: Weight, 360 gms. Muscle pale, not firm; hypertrophy and dilatation of left ventricle. Associated pathological findings: Chronic diffuse nephritis.

Autopsy No. 2627. Black, age 59. Clinical diagnosis: Hemiplegia; arteriosclerosis. Position of tumor: One subperitoneal. Size of tumor: Fist. Heart condition: Weight, 345 gms. Hypertrophy; sclerotic aortic and mitral valves. Associated pathological findings: Chronic interstitial nephritis; cerebral hæmorrhage; general arteriosclerosis.

Autopsy No. 2660. White, age 59. Clinical diagnosis: Cardiac hypertrophy and dilatation; mitral regurgitation. Position of tumor: Few interstitial. Size of tumor: Small. Heart condition: Weight, 580 gms. Hypertrophy and dilatation; pale muscle.

Autopsy No. 2674. Black, age 44. Clinical diagnosis: Valvular heart disease; chronic nephritis; anasarca. Position of tumor: several interstitial and submucous. Size of tumor: small. Heart condition: Weight, (?) gms. Hypertrophy and dilatation; sclerotic valves and coronary arteries. Associated pathological findings: Arteriosclerosis; nutmeg liver; cyanotic induration of kidneys.

Autopsy No. 2681. White, age 38. Clinical diagnosis: Hemiplegia; status epilepticus. Position of tumor: Few interstitial. Size of tumor: Very small. Heart condition: Weight, (?) gms. Chronic myocarditis; sclerotic aortic and mitral valves. Associated pathological findings: Chronic interstitial nephritis; syphilitic liver.

Autopsy No. 2692. White, age 57. Clinical diagnosis: Pneumonia; myocarditis. Position of tumor: One interstitial. Size of tumor: Small. Heart condition: Weight, 356 gms. Normal; sclerosis of aorta. Associated pathological findings: Pneumonia; chronic interstitial nephritis.

Autopsy No. 2697. Black, age 55. Clinical diagnosis: Sarcoma of femur; myocardial degeneration. Position of tumor: One subperitoneal, calcified. Size of tumor: Very large. Heart condition: Weight, 375 gms. Small and firm; small sarcomatous nodule. Associated pathological findings: Sarcoma of femur.

Autopsy No. 2699. White, age 79. Clinical diagnosis: Apoplexy; pneumonia; arteriosclerosis. Position of tumor: One subperitoneal polyp. Size of tumor: 3 x 5 cm. Heart condition: Weight, 260 gms. Chronic interstitial myocarditis; sclerosis of mitral and aortic valves and coronary arteries. Associated pathological findings: General arteriosclerosis; chronic interstitial nephritis; cerebral œdema.

Autopsy No. 2749. White, age 61. Clinical diagnosis: Pneumonia; meningitis; valvular disease of heart. Position of tumor: One subperitoneal, calcified. Size of tumor: Baseball. Heart condition: Weight, 340 gms. Dilatation; chronic mitral and aortic sclerosis; calcareous coronaries. Associated pathological findings: general arteriosclerosis.

Autopsy No. 2753. Black, age 58. Clinical diagnosis: Cerebral thrombosis; arteriosclerosis. Position of tumor: Several submucous. Size of tumor: Small. Heart condition: Weight, 300 gms. Slight chronic myocarditis. Associated pathological findings: Chronic parenchymatous nephritis; old cerebral hæmorrhage.

Autopsy No. 2760. Black, age 61. Clinical diagnosis: Meningo-myelitis. Position of tumor: One interstitial. Size of tumor: Small. Heart condition: Weight, 205 gms. Sclerosis of valves and coronary arteries; syphilitic aortitis. Associated pathological findings: Chronic interstitial nephritis.

Autopsy No. 2766. Black, age 50. Clinical diagnosis: Tubercular meningitis. Position of tumor: Few. Size of tumor: Small. Heart condition: Weight, 450 gms. Cloudy swelling; mitral sclerosis. Associated pathological findings: General arteriosclerosis; chronic interstitial nephritis; diffuse meningeal hæmorrhage.

Autopsy No. 2768. White, age 53. Clinical diagnosis: Diabetes mellitus. Position of tumor: One submucous. Size of tumor: Lemon. Heart condition: Weight, 310 gms. Brown atrophy; chronic myocarditis; slight mitral, aortic and coronary sclerosis. Associated pathological findings: (?)

Autopsy No. 2773. Black, age 53. Clinical diagnosis: Tuberculosis of lungs; pelvic tumor. Position of tumor: Multiple; calcified. Size of tumor: Largest, Two fists. Heart condition: Weight, 370 gms. Chronic myocarditis; fatty myocardium; sclerosis of mitral and aortic valves, and aorta and coronaries. Associated pathological findings: Œdema of lungs; chronic interstitial nephritis.

Autopsy No. 2780. Black, age 30. Clinical diagnosis: Hæmophilia; valvulitis. Position of tumor: Few interstitial nodules. Size of tumor: Small. Heart condition: Weight, 350 gms. Chronic fibroid; myocarditis. Associated pathological findings: Hypernephroma of left kidney.

Autopsy No. 2781. White, age 52. Clinical diagnosis: Cancer of liver; tuberculosis of lungs. Position of tumor: One subperitoneal. Size of tumor: 1 cm. Heart condition: Weight, 220 gms. Fatty degeneration. Associated pathological findings: Healed tuberculosis of lungs; cancer of gallbladder.

Autopsy No. 2818. Black, age 53. Clinical diagnosis: Cardiac dilatation; myocarditis. Position of tumor: Multiple. Size of tumor: Largest, 20 x 17 cm. Heart condition: Weight, 230 gms. Valves normal; slight sclerosis of coronaries; myo-

cardium pale and flabby. Associated pathological findings: Mixed tumor of carotid with metastases to liver; chronic passive congestion of kidneys.

Autopsy No. 2826. White, age 49. Clinical diagnosis: Mitral and tricuspid regurgitation. Position of tumor: Two nodules. Size of tumor: Pea and acorn. Heart condition: Weight, 460 gms. Chronic pericarditis; hypertrophy; chronic mitral and aortic sclerosis. Associated pathological findings: Chronic parenchymatous nephritis.

Autopsy No. 2856. White, age 74. Clinical diagnosis: Myocardial degeneration, arteriosclerosis. Position of tumor: Two subperitoneal. Size of tumor: Small. Heart condition: Weight, 410 gms. Dilatation of right heart. Associated pathological findings: General arteriosclerosis.

Autopsy No. 2858. White, age 67. Clinical diagnosis: Senile dementia; pneumonia. Position of tumor: One nodule. Size of tumor: 3 cm. Heart condition: Weight, 360 gms. Cloudy swelling. Associated pathological findings: General arteriosclerosis.

Autopsy No. 2860. Black, age 38. Clinical diagnosis: Myocarditis; mitral regurgitation; chronic parenchymatous nephritis. Position of tumor: Two subperitoneal calcified. Size of tumor: 8 cm. Heart condition: Weight, 500 gms. Hypertrophy and dilatation; chronic aortic endocarditis. Associated pathological findings: Cirrhosis of liver; chronic parenchymatous nephritis.

Autopsy No. 2869. White, age 48. Clinical diagnosis: Status epilepticus. Position of tumor: One submucous polyp. Size of tumor: 3½ cm. Heart condition: Weight, 480 gms. Chronic endocarditis; hypertrophy; atheroma of aorta. Associated pathological findings: Chronic diffuse nephritis; tuberculosis of lungs.

TABLE II

SHOWING VARIOUS HEART LESIONS FOUND ON POST-MORTEM EXAMINATION

	Fibroids, 100 cases	Other Conditions, 100 cases
Normal hearts	12	18
Sclerotic mitral valves	57	44
Sclerotic aortic valves	45	45
Sclerotic tricuspid valves	9	3
Sclerotic pulmonic valves	2	..
Chronic pericarditis	8	2
Chronic myocarditis	15	14
Chronic endocarditis	2	2
Epicardial plaques	5	6
Sclerosis of coronaries	14	7
Atheroma of aorta	2	19
Hypertrophy	34	29
Dilatation	9	21
Fatty heart	14	18
Brown atrophy	7	3
Cloudy swelling	2	..
Aneurism	1	5
Atrophy	2	..
Acute endocarditis	1	..
Acute pericarditis	2	4
Acute aortic disease	1	2
Acute mitral disease	..	1
Acute tricuspid disease	..	1
Hydropericardium	6	1
Hæmopericardium	..	1
Pericardial ecchymosis	1	..
Subendocardial hæmorrhage	..	1
Œdema of the myocardium	1	..
Myxoma of the heart	1	..
Lipomatosis pericardii	..	3
Total lesions	241	232

TABLE III

(Age, from twenty to twenty-nine years)

	Fibroids, 2 cases	Other Conditions[1]
Normal	2	..
Acute endocarditis	..	1
Sclerosis of coronaries	..	1
Total lesions	2	2

[1] Multiple neuritis; gangrene of lung.

TABLE IV

(Age, from thirty to thirty-nine years)

	Fibroids, 13 cases	Other Conditions[1]
Normal	5	8
Hypertrophy	4	1
Dilatation	2	..
Chronic mitral disease	5	1
Chronic aortic disease	2	1
Sclerosis of coronaries	2	..
Hydropericardium	2	..
Sclerosis of tricuspid	2	..
Pericardial ecchymosis	1	..
Chronic pericarditis	1	..
Acute pericarditis	1	..
Chronic myocarditis	2	2
Epicardial plaques	1	1
Chronic endocarditis	1	..
Fatty heart	1	1
Atrophy of heart	1	..
Atheroma of aorta	..	1
Total lesions	28	8

[1] Cerebral tumor, pyosalpinx, tuberculous meningitis, miliary tuberculosis, gall-stones, pulmonary tuberculosis, uræmia, cancer of breast, syphilis, cancer of rectum, gastric ulcer, pneumonia.

TABLE V

(Age, from forty to forty-nine years)

	Fibroids, 25 cases	Other Conditions[1]
Normal	7	5
Dilatation	3	4
Hypertrophy	7	5
Chronic aortic disease	11	8
Chronic mitral disease	9	13
Fatty heart	3	3
Epicardial plaques	1	1
Chronic myocarditis	4	3
Chronic pericarditis	1	2
Chronic endocarditis	1	1
Sclerosis of coronaries	1	2
Cloudy swelling	1	..
Aneurism	1	2
Hydropericardium	1	..
Small heart	1	..
Atheroma of aorta	..	4

Acute aortic disease	..	1
Acute tricuspid disease	..	1
Brown atrophy	..	1
Acute pericarditis	..	1
Hæmopericardium	..	1
Total lesions	45	53

[1] Cancer of pancreas, chronic parenchymatous nephritis, pulmonary tuberculosis, acute nephritis, apoplexy, aortic valvular disease, pneumonia, miliary tuberculosis, aortic aneurism, pelvic peritonitis, diabetes, acute pericarditis, pachymeningitis, purpura, tabes dorsalis, cancer of uterus, erysipelas.

TABLE VI

(Age, from fifty to fifty-nine years)

	Fibroids, 27 cases	Other Conditions[1]
Normal	..	2
Hypertrophy	10	8
Dilatation	1	12
Chronic mitral disease	17	9
Chronic aortic disease	14	12
Chronic tricuspid disease	1	1
Sclerosis of coronaries	4	2
Epicardial plaques	2	3
Chronic pulmonic disease	1	..
Hydropericardium	1	1
Fatty heart	4	6
Brown atrophy	2	2
Myxoma of heart	1	..
Atheroma of aorta	1	4
Chronic pericarditis	3	..
Chronic myocarditis	3	5
Œdema of myocardium	1	..
Acute pericarditis	..	1
Aneurism	..	1
Total lesions	66	67

[1] Pernicious anæmia, meningomyelitis, chronic interstitial nephritis, pneumonia, myocarditis, adiposa dolorosa, tabes dorsalis, aneurism, pulmonary tuberculosis, uræmia, pachymeningitis, apoplexy, cancer of vagina, valvular heart disease, myxœdema, septic cholangeitis.

TABLE VII

(Age, from sixty to sixty-nine years)

	Fibroids, 17 cases	Other Conditions[1]
Normal	..	1
Hypertrophy	6	8
Dilatation	2	3
Fatty heart	5	4
Chronic mitral disease	13	13
Chronic aortic disease	6	13
Chronic tricuspid disease	4	1
Sclerosis of coronaries	2	3
Atheroma of aorta	1	5
Chronic myocarditis	2	3
Hydropericardium	1	..
Chronic pericarditis	3	2
Brown atrophy	3	..
Cloudy swelling	1	..
Epicardial plaques	..	1
Chronic endocarditis	..	1
Lipomatosis pericardii	..	3
Acute mitral disease	..	1
Acute aortic disease	..	1
Total lesions	49	62

[1] Chronic interstitial nephritis, fractured thigh, cancer of vagina, emphysema of lungs, cancer of stomach, pneumonia, apoplexy, gall-stones, senile dementia, rheumatism, valvular heart disease, abscess of kidney.

TABLE VIII

(Age, from seventy to seventy-nine years)

	Fibroids, 14 cases	Other Conditions[1]
Hypertrophy	7	7
Dilatation	1	2
Chronic mitral disease	12	8
Chronic aortic disease	11	10
Chronic tricuspid disease	2	..
Chronic pulmonic disease	1	..
Sclerosis of coronaries	3	..
Acute aortic disease	1	..
Epicardial plaques	1	..
Chronic myocarditis	4	1
Chronic pericarditis	1	..
Fatty heart	1	4
Brown atrophy	2	..
Hydropericardium	1	..
Atheroma of aorta	..	4
Aneurism of aorta	..	2
Subendocardial hæmorrhage	..	1
Total lesions	48	39

[1] Pneumonia, chronic parenchymatous nephritis, cancer of stomach, senility, pulmonary tuberculosis, uræmia, aneurism of aorta, abscess of kidney, chronic interstitial nephritis.

TABLE IX

(Age, eighty years and over)

	Fibroids, 1 case	Pneumonia
Chronic aortic disease	1	1
Sclerosis of coronaries	1	..
Chronic tricuspid disease	..	1
Atheroma of aorta	..	1
Total lesions	2	3

TABLE X

(Age, twenty to thirty-nine years)

	Fibroids, 28 cases	Other Conditions[1]
Normal	9	10
Hypertrophy	8	2
Dilatation	4	1
Chronic mitral disease	9	4
Chronic aortic disease	6	4
Sclerosis of coronaries	6	1
Sclerosis of tricuspid	3	..
Hydropericardium	3	..
Pericardial ecchymosis	1	..
Chronic pericarditis	1	..
Acute pericarditis	2	..
Chronic myocarditis	6	..
Epicardial plaques	4	8
Chronic endocarditis	3	1
Fatty heart	1	1
Atrophy of heart	1	5
Atheroma of aorta	2	..
Cloudy swelling	..	2
Subendocardial hæmorrhage	..	1
Lipomatosis pericardii	..	1
Acute endocarditis	1	1
Total lesions	61	32

[1] Miliary tuberculosis, gall-stones, cancer of breast, cancer of rectum, gastric ulcer, cerebral tumor, tuberculous meningitis, ovarian cyst, pulmonary tuberculosis, cancer of liver, syphilis, uræmia, pneumonia, pyosalpinx, multiple neuritis, gangrene of lung.

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A STUDY OF THE COMPARATIVE VALUE OF FUNCTIONAL TESTS IN THE SURGICAL DISEASES OF THE KIDNEY SECONDARY TO OBSTRUCTION IN THE LOWER URINARY TRACT

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In our previous communications[2] on the subject of renal function attention has been called to the value of phthalein in affording an index to the renal functional capacity in the changes resulting from obstruction in the lower urinary tract. It was pointed out that such patients are frequently the subject of hydronephrosis, pyelonephritis, pyonephrosis, pressure atrophy, and resulting changes in functional activity, and that the urine output, urea, and total solids might be practically normal and yet the patient be on the verge of renal failure which would surely be precipitated by any radical surgical interference. The phthalein was shown to be of value in all these cases and to allow of differentiation between those cases with severe renal damage and those in which the renal involvement was slight. It was further shown that the marked improvement of the renal condition and operative risk which followed the adoption of Young's preliminary treatment (constant catheter and forcing of water) was the result to a large extent of the improvement of the renal functional capacity. It was claimed that the test made it possible to determine the cases suitable or unsuitable for operation and also to determine in any given case the most propitious time for surgical intervention.

In this study a number of tests have been utilized in order to determine if any individual test or any combination of tests furnishes information other than that derived from the use of phthalein. The number of functional tests has increased to such an extent that it is essential to determine which can be discarded without loss. Only through familiarity with the reliability, value, limitations, peculiarities, and the significance of the findings of each test in the various types of disease is the most profitable selection of tests made possible.

Renal functional capacity is usually ascertained in one of two ways: (1) Tests of excretory capacity through the quantitative determinations of the secretion of various substances in the urine,— dyes, methylene blue, indigo-carmin, rosaniline, and phthalein; other chemicals, potassium iodide, lactose, salicylates, sodium chloride, urea, sugar following phloridzin, and the enzyme, diastase. (2) Tests of retention through the determination of the concentration of certain substances in the blood, ions — through electrical conductivity, molecules and ions — through cryoscopy, and urea, total incoagulable nitrogen and cholesterin. Choice has been made of what has appeared to be the most important representatives of these groups; viz., of the first, phthalein, lactose, diastase, and urea; of the second, cryoscopy and blood urea.

THE TESTS EMPLOYED AND THEIR TECHNIQUE

1. The *phthalein* was used according to the usual technique.

2. *Lactose* was employed according to Schlayer's technique:[3] 2.5 gms. of lactose are dissolved in 25 ccm. of freshly distilled water, placed in a 50 ccm. Erlenmeyer flask stoppered with cotton. This is pasteurized for 4 hours on four successive days between 75°–80° C., and is injected intravenously, the number of hours necessary for excretion being determined. Nylander's and Fehling's tests were used to demonstrate the presence or absence of sugar. No quantitative polarimetric determinations which are advised by Schlayer were utilized, since only in a small proportion of cases did glycosuria occur.

3. *Diastase.* Diastase was introduced into functional renal work by Wohlgemuth[4] for

[2] Rowntree and Geraghty: J. Phar. & Exp. Therap., 1910, i, 579; Arch. Internal Med., 1912, ix, 284.

[3] Schlayer and Takayasu: Deutsche Arch. f. klin. Med.

[4] Wohlgemuth: Ztschr. f. Urol., 1911, v, 801.

[1] From the Pharmacological and Physiological Chemical Laboratories of the Johns Hopkins University and the Genito-Urinary Clinic of the Johns Hopkins Hospital.

determining the relative functional capacity of the two kidneys. A modification of this method was devised by us to make the test adaptable for determining total renal functional capacity. The technique which we employed was elaborated in conjunction with Dr. Thomas R. Brown,[1] who has demonstrated with it that the total daily output in urine is fairly constant. The technique employed was as follows: One-fifth of the twenty-four hour urine collected under toluol was neutralized and diluted to 1 L. By means of a graduated 2 ccm. pipette, decreasing amounts of urine were placed in a series of twelve tubes (arranged in a rack) as follows: 1.8, 1.6, 1.4, 1.2, 1, 0.8, 0.6, 0.4 ccm. Ten ccm. of the diluted urine was further diluted to 20 ccm. and of this decreasing amounts placed in the remaining tubes of the series as follows: 0.4, 0.2, 0.1, 0.05 ccm. To each tube was added sufficient 1 per cent NaCl solution to bring the total volume in each tube to 2 ccm. Two ccm. of a 0.1 per cent freshly prepared soluble starch solution was added to each tube. The rack with the tubes was placed in a water-bath at 38° C. for a half-hour, then transferred to cold water for 3 minutes. Sufficient 1/50 N iodine solution was added drop by drop in amounts sufficient to elicit a permanent color. The occurrence of blue or violet shows incomplete digestion, while the last tube without the violet color indicates the diastatic activity of the urine and from it is calculated. The diastatic activity is expressed by $d\,\frac{30'}{30^{\circ}}$ which represents the number of ccm. of 0.1 per cent starch solution capable of being digested at 38° C. in 30 minutes by 1 ccm. of the urine as originally diluted.

The *urea* content of the twenty-four hour specimens of urine was determined by the Doremus and the Marshall[2] methods, the results of each being given.

The principle of Marshall's method consists in the conversion of the urea into ammonium carbonate by means of an enzyme, urease, present in an extract of the soy bean, and the titration with standard hydrochloric acid and methyl orange. "Two 5 ccm. portions of urine are measured into flasks of 200–300 ccm. capacity and diluted with distilled water to about 100–125 ccm. Two ccm. of the enzyme solution are added to one flask, a few drops of toluol to each and the solutions allowed to remain, well stoppered, at room temperature over night. The fluid in each flask is titrated with 0.1 NHCl and methyl orange. The amount of hydrochloric acid required for neutralization of the contents of the flask containing the urine and enzyme solution less the amount required for neutralization of 5 ccm. urine alone and 2 ccm. enzyme solution alone corresponds to the urea originally present in the urine. This value multiplied by 0.6 gives the number of grams of urea per liter of urine.

The freezing point of serum was made in the ordinary way, utilizing the Beckmann apparatus, 25 to 50 ccm. of blood usually being taken from the patient.

The blood urea was determined according to Marshall's method[3] which is as follows: Two equal portions of the serum (3 ccm. or 5 ccm.) are measured into two small test tubes, 1 ccm. of the enzyme solution added to one, toluol to each, and the tubes allowed to remain over night. The contents of each tube are washed into tall cylinders, alcohol and sodium carbonate added, and the ammonia removed by means of a strong air current. The ammonia is collected in flasks containing 25 ccm. N/50 hydrochloric acid. When all the ammonia has been driven over, the excess of acid in the flasks is titrated with N/50 sodium hydroxide and sodium alizarin sulphonate. The amount of hydrochloric acid required to neutralize the ammonia from the serum and enzyme solution less the amount required for the ammonia from the serum alone represents the urea present in the amount of serum taken.

THE RESULTS OBTAINED

In the following table are presented data relating to 43 cases, in the majority of which a combination of all of the tests mentioned has been utilized. The cases for the most part exhibited varying degrees of urinary obstruction.

Our earlier views in regard to the value of the phthalein test in this type of case require

[1] To appear shortly.

[2] Marshall: J. Biol. Chem., 1913, xiv, 283.

[3] To appear in an early number of Journal Biological Chemistry.

	Surg. No. Case	Name	Date admitted	Diagnosis	B. P.	Date	Phthalein Appear Minutes	Phthalein 1st Hr.	Phthalein 2d Hr.	Diastase $d\frac{38°}{30'}$	24 Hour Amount ccm.	Urine Urea grams	Cryoscopy	Lactose	Remarks
1	30731	M. L. B. 60 years	11/12/12	Enlarged prostate Cystitis			17	9%	13%	5.0	3100			Negative	Prostatectomy. Good recovery.
2	30765	W. H. A. 70 years	11/12/12	Hypertrophy prostate			15	17%	20%					Negative	Prostatectomy. Recovery.
3	30901	Dr. J. K. 63 years	11/16/12	Hypertrophy prostate	185	11/4/12	7	22%	5%					Negative	Prostatectomy. Recovery.
4	30919	J. S. J. 59 years	11/19/12 1/2/13	Hypertrophy prostate Chr. nephritis	220	11/16/12 1/1/13 1/21/13 1/23/13	14 62? 14 15	6% 10% 21% 17%	8% 20% 20% 30%				.62 1/2/13	Negative 1/2/13	Prostatectomy. Recovery.
5	30928	Wm. W. 69 years	11/19/12 11/30/12	Enlarged prostate Pyelonephritis	204	11/17/12 11/30/12	30 20	trace 15%	5% 10%	11/21/12 10.0	2900		.59 11/21/12	Negative 11/20/12	Prostatectomy. Died hæmorrhage.
6	30918	Wm. C. 55 years	11/16/12 11/24/12	Tabes 200 ccm. resid. urine	188	11/16/12 11/24/12	11 15	45% 48%	11% 12%	2.5	1400			Negative 11/20/12	No operation.
7	30932	G. F. 17 years	11/18/12 11/24/12 1/27/13 2/7/13	Double hydronephrosis Congenital valves Prostatic urethra		11/18/12	? ? ? ?	trace trace trace trace	5% trace 3% 3%	11/22/12 2.0 2/7/13 0	3340 2050	5.0 14.0	.61 11/20/12 .63 1/27/13 .66 2/7/13	Negative 11/20/12	Several diastase estimations of urine between 2/6–12/13 all same. Blood urea, 1.332 gms. per liter. Died pneumonia.
8	30962	C. W. 64 years	11/21/12 12/7/12	Hypertrophy prostate Operation	Put	11/21/12 11/24/12 on forced	22 13 water.	32% 25% Toxic	16% 35%				.55 11/22/12 .48 12/7/12 .54 12/12/12	4 hrs.[1]	Prostatectomy. Recovery.
9	30990	C. R. 80 years	11/26/12 12/9/12 1/1/13 3/18/13	Hypertrophy prostate Cystitis Pyelonephritis, Very weak Condition bad	170	11/27/12 12/9/12 1/1/13	22 64? 27	trace 5% 17%	5% 6% 15%	2.0 1.7	2980 3/11/13 1580	20.8 18.1 *15.3*	.57 1/2/13		Prostatectomy. Recovery.
10	30997	J. C. 62 years		Hypertrophy prostate	165	11/27/13	18	42%	20%					Negative 11/27/12	Prostatectomy. Recovery.
11	31028	F. E. 58 years	12/3/12 12/9/12	Hypertrophy prostate Arteriosclerosis	225	12/3/12	12	30%	20%	12/7/12 5.0	3400 12/6/12	10.2	.57 12/5/12	Negative 12/5/12	Prostatectomy. Recovery.
12	31027	J. B. 34 years	12/3/12	Nephroptosis, left	144	12/3/12	5½	48%	17%				.58 12/5/12	Negative 12/5/12	Suspension. Recovery.
13	31029	S. B. 77 years	12/3/12	Hypertrophy prostate Arteriosclerosis	220	12/3/12	12	28%	10%	12/4/12 2.5	2300 12/3/12	15.8	.51 12/7/12	Negative 12/5/12	Prostatectomy. Died broncho-pneumonia.
14	31039	M. E. C. 57 years	12/4/12	Hypertrophy prostate		12/4/12	15	17%	14%				.53 12/5/12		Prostatectomy. Recovery.
15	31059	Jos. P. 75 years	12/6/12	Hypertrophy prostate Ves. calculi Suprapubic drainage		12/7/12 1/1/13 1/21/13	32 62 14	7% 10% 24%	12% 20% 20%	12/13/12 2.5	3220 12/12/12		.60 12/12/12 .71 1/2/13 .59 1/23/13	Negative 1/23/13	Prostatectomy, 1/23/12. Died suddenly after going home.
16	31164	N. C. 75 years	12/23/12	Cancer bladder with partial retention.		12/23/12	7	22%	15%				.51 12/23/12		No operation.
17	31214	E. J. 60 years	1/1/13 1/7/13	Arteriosclerosis, no retention of urine Chr. nephritis	200	1/1/13	13	10%	9%	1/8/13 5.0	1625 1/7/13 2200 1/2/13 2760 1/3/13	17.9	.59 .56	Negative 1/23/13	No operation.

[1] Not quantitative test. 11/22/12.

18	31225	D. M. D. 65 years	1/2/13 1/8/13	Double polycystic kidneys	high	1/1/13	20	trace	trace	1/8/13 3.3	2650	18.5	.70 1/2/13		Died suddenly a few weeks later. No autopsy.
19	31249	J. S. 53 years	1/4/13	Hypertrophy prostate		1/4/13	9	34%	14%				.56 1/9/13		Prostatectomy. Recovery.
20	31313	L. F. 72 years	1/15/13	Hypertrophy prostate Bad heart condition		1/15/13 1/21/13	16 10	17% 17%	14% 15%				.56 1/16/13		Prostatectomy. Recovery.
21	29252	L. R. 45 years	1/18/13	Bilateral pyonephrosis Condition bad		1/18/13	35	trace	trace				.61 1/20/13		No operation.
22	31399	J. T. C. 74 years	2/2/13	Hypertrophy prostate		2/2/13	23	7%	1%				.58 2/1/13		Prostatectomy. Recovery.
23	31573	S. B. R. 42 years	2/22/13	Contracture vesical neck		2/22/13	10	45%	13%		3/10/13 1580	*15.3*	.54 2/24/13		Punch. Recovery.
24	31553	Col. F. M. 72 years	2/20/13	Cancer prostate Paralysis, Myocarditis		2/20/13 3/6/13	13 12	30% 41%	10% 17%	2/28/13 2.5	1560 2/27/13	11.7 *10.4*	.54 2/26/13		Prostatectomy, 3/7/13. Recovery.
25	31592	Wm. L. 63 years	2/25/13	Hypertrophy prostate						10.0	2380	19.0 *15.8*	.54 2/26/13		Prostatectomy, 2/28/13. Recovery.
26	31546	Gen. J. S. 71 years	2/19/13	Chr. cystitis Contracted bladder	140	2/23/13 3/6/13	6 10	20% 26%	15% 15%	2/28/13 3.3	1220 2/27/13	19.5 *15.9*			No operation.
27	31593	L. V. M. 69 years	2/26 13	Hypertrophy prostate Acute double pyelone-phritis		3/3/13	10	22%	19%	3/5/13 3.3	830 3/2/13	9.5 *8.7*	.60 3/1/13		Prostatectomy, 3/5/13. Blood urea, 0.660 gms. per liter. Died.
28	31620	Col. C. W. 75 years	3/1/13	Hypertrophy prostate		3/2/13 3/4/13 3/6/13	10 11 10	22% 30% 38%	17% 18% 28%	3/5/13 2.5	5080 3/5/13	44.8 *26.4*	.50 3/4/13		Prostatectomy, 3/7/13. Recovery.
29	31619	Dr. A. H. L. 72 years	3/1/13	Cancer prostate		3/3/13	13	27%	10%				.55 3/4/13		Prostatectomy, 3/5/13. Blood urea, .426 gms. per liter. Recovery.
30	31657	E. C. B. 69 years	3/1/13	Hypertrophy prostate Suprapubic for drainage 3/14/13	140	3/5/13 3/8/13 3/10/13	41 35 35	3% 14% 17%	10% 15%	3/11/13 1.3 3/18/13 2.0	4585 3/11/13 5480 3/18/13 5025 3/19/13	*21.9* 35.6 *30.2*	.54 3/10/13		
31	31670	G. N. 66 years	3/7/13	Cancer prostate	140	3/8/13	14	48%	17%	3/12/13 3.3	1555 3/12/13	13.9 *11.9*	.61 3/11/13		Prostatectomy operation. Blood urea, .470 gms. per liter. Recovery.
32	31705	R. F. 71 years	3/12/13	Cancer prostate		3/9/13	11	33%	27%	3/19/13 2.5	2320 3/19/13	23.3	.60 3/19/13		Prostatectomy, 4/2/13. Blood urea, .222 gms. per liter. Recovery.
33	31782	W. H. D. 48 years	3/24/13	Hæmaturia		3/22/13 3/25/13 3/26/13	10 10 2½	38% 43% 1st. 15 min.	12% R.15 L.18	1.7	1830 3/24/13	6.4 *11.9*	.59 3/25/13	Negative 3/25/13	No operation. Blood urea, .230 gms. per liter.
34	31824	C. K. 65 years	3/28/13	Hypertrophy prostate		3/30/13	19	trace	trace	4.8 3.3	1575 4/9/13	9.5 *4.4*	.61 .59 4/8/13	Negative 4/9/13	Prostatectomy.
35	29319	J. T.	2/23/12	Cancer prostate	155	2/26/12	9	15%	18%					8 hours	
36	31779	G. H. 35 years	3/21/13	Double polycystic kidney	150	3/28/13	10	20%	16%	1.7	1260 4/2/13 800 4/9/13	12.6 4.4			Went home.

	Surg. No. Case	Name	Date admitted	Diagnosis	B. P.	Date	Phthalein			Diastase $d\frac{38°}{30'}$	24 Hour Amount cc.	Urine Urea grams	Cryoscopy		Lactose	Remarks
							Appear minutes	1st Hr.	2d Hr.							
37	31890	A. H. 53 years	4/6/13	Hypertrophy prostate		4/8/13 4/20/13	33 14	16% 11%	11% 8%	3.3	1860 4/12/13	*15.4*	.61	4/9/13	4/9/13*	Blood urea, .350 gms. per liter. 4/9/13.
38	31878	J. F. N. 59 years	4/4/13	Hypertrophy of prostate	115	4/10/13	10	trace	7%				.52	4/19/13		Blood urea, 2.17 gms. per liter. 4/19/13. Sent home.
39		B.	5/15/13	Bilateral pyelonephritis			12	12%	18%		4000	4.0			Negative	Iodid 48 hours.
40	32020	J. F. 61 years	4/23/13	Hypertrophy prostate		4/21/13 4/27/13	20 12	11% 25%		5.0	4335 4/29/13	*15.2* 13.0	.59	4/29/13	1st hr. pos 9th & 10th pos.	Prostatectomy, 5/2/13. Blood urea, .198 gms. per liter. Recovery.
41	32048	L. D. 70 years	4/28/13	Carcinoma prostate Vesical calculus		4/29/13 5/2/13	16 10	23% 40%	10% 14%				.57		1st & 2d hrs. pos. 5th & 6th pos.	Prostatectomy, 5/2/13. Blood urea, .450 gms. per liter. Recovery.
42	31998	J. L. 53 years	4/21/13	Carcinoma prostate Resid. urine, 450 cc.	170	4/21/13 4/27/13	55 16	12% 34%	5% 8%	5.0	2040 4/30/13	*19.88*	.54	4/29/13	1st 4 hrs. pos.	Prostatectomy, 5/2/13. Recovery. Blood urea, .350 gms. per liter.
43	Not indexed	H. 55 years	6/1/13	Vesical calculi stricture		5/22/13 5/28/13		trace trace	trace trace				.60 .66			Blood urea, 1.50 gms. per liter. 2.64 gms. per liter. Dead.

Urea in urine indicated by *italics* was determined by Marshall's method.
* First hour negative; second hour positive; third and fourth hours positive; other eight negative.

no revision. As an index of the total renal function it has proven uniformly accurate and reliable.

The lactose test has been employed in 22 cases. In all of the earlier cases the urine was collected for 4 hours and each 2 hours thereafter up to 12 hours following Schlayer's technique. In only two cases, Nos. 8 and 35, was a positive test for sugar obtained. In No. 6, a case of tabes, with 200 ccm. residual urine, the phthalein showed practically a normal renal function, while clinical findings showed no evidence of renal disease. However, no lactose was obtained in this case. In Case 10, with hypertrophied prostate and normal renal function as indicated by phthalein, no lactose was excreted. This patient was in good clinical condition, and successful prostatectomy was performed. In four later cases in which following the lactose injection the urine was collected at hourly intervals, a trace of lactose was obtained in some of the periods. The renal function in these cases was more impaired than in many of the earlier ones in which the first collection was for 4 hours. It is not improbable that if the urine had been collected at hourly intervals in these earlier cases, a positive test might have been obtained in some of the hourly specimens in many of the cases which showed a negative finding with the technique employed. In two of the four cases mentioned above the excretion was complete within 4 hours. One of these, however, had a severe grade of renal involvement.

The majority of the patients in this group were on forced water and excreted large quantities of urine. Marked dilution resulting from this polyuria may help to explain the failure to detect lactose where traces only were excreted. The failure on the part of lactose to appear in the urine is in keeping with our earlier experiences with phloridzin in this type of renal disease. Phloridzin was used in eight patients suffering from renal changes secondary to prostate hypertrophy but in not a single instance was sugar recovered. Further observations were therefore not made.

The lactose test in this type of case has proven of but little value from a diagnostic

standpoint, and of no value in relation to prognosis.

Twenty-seven diastase estimations were made in 24 cases. We have considered the normal diastatic activity of the diluted urine as $d\,\frac{38°}{30'}=5.0$.

In some cases with severe renal damage, the diastase content was normal. A striking example of this is Case 5, in which the phthalein and the clinical condition indicated severe renal impairment. The diastase value was $d\,\frac{38°}{30'}=10$. This patient died of hæmorrhage following prostatectomy, and at autopsy a well marked chronic diffuse nephritis with chronic bilateral pyelonephritis and a moderate grade of hydronephrosis was discovered. One kidney was one-fourth the size of the other, the larger kidney weighing 170 grams. The character of the lesions was similar in both kidneys. On the other hand, in another case, Case 18, in slight hæmaturia with advanced double polycystic disease of the kidneys, with a blood pressure of over 200 mm., a trace of phthalein in two hours, and a freezing point of −.70°, the diastase was only slightly reduced being $d\,\frac{38°}{30'}=3.3$. This patient died a few weeks after leaving the hospital, without the cause of death being ascertained.

In Case 33 in which the phthalein and clinical findings showed only slight renal impairment, the diastase was markedly decreased, $d\,\frac{38°}{30'}=1.7$.

In the majority of instances of disease a decreased diastatic activity is present, but no constant or definite relationship between the diastatic activity and the true renal function as shown by phthalein and clinical findings exists. Where, however, a complete absence of diastase in the urine occurs, it apparently indicates a bad prognosis. Two cases, Nos. 7 and 43, with severe renal impairment as shown by excretory tests and the presence of marked cumulative phenomena, showed no diastase, and death occurred within two weeks in Case 43 and within one month in the other. Autopsies were obtained in each of these cases and revealed advanced renal disease.

Urea estimations of the twenty-four hour output were made in twenty-two cases. These give information of but little value. Patients with very low renal function and advanced disease frequently excrete larger amounts than those in good clinical condition and with comparatively good renal function. When, however, there is a persistently very low urea elimination, it indicates a severe grade of renal disturbance.

In utilizing the second group of tests, viz., tests of retention, a consideration of what is to be accepted as the normal limit seems advisable. Although Folin and Denis [1] accept 0.28 gm. urea per liter as the normal, where no dietary control is exercised in health or in many diseases without recognizable renal changes much higher values are encountered. From our observations it seems advisable to place the limit of normal at 0.55 for urea, and 0.50 gms. per L. total incoagulable N and −.60 for the freezing point. Evidences of retention greater than this are referred to as cumulative phenomena.

Cryoscopy has been employed in 34 cases, 43 determinations having being made. Variations from −0.48 to −0.71 were encountered. In every instance except one a normal phthalein has been associated with a normal freezing point. In this case, No. 31, δ was −0.61 with a phthalein practically normal, 48 per cent for 1 hour. The urea was close to the higher limit of normal, 0.47 gm. per liter.

The highest δ (−0.48) occurred in a man who was being continuously forced to drink large quantities of water.

Nine cases showed cumulative phenomena in that they had a δ lower than −0.60. Except in the case, No. 31, already mentioned, renal function was seriously involved in each instance, as indicated by phthalein and the general clinical condition.

A decrease in phthalein output of serious import may be associated with a normal δ. In a group of 4 cases, Nos. 5, 22, 38, and 43, a trace or less than 10 per cent phthalein was excreted in 2 hours, but the freezing points were normal. These cases were all seriously ill clinically and in the only two cases, 38 and 43, in which blood urea was determined, it was found to be abnormally high, 2.17 and 1.50

[1] Folin and Denis: J. Biol. Chem., 1913, xiv, 29.

gms. per L. respectively. Repetition of tests 5 days later showed in Case 43 a δ −0.66 and a urea 2.64 gm. per L. This patient died in uræmia, while in the other case operation was refused and the patient sent home on account of his poor general condition.

Blood urea was determined in 12 cases. In every instance in which the phthalein was high the blood urea was normal. Abnormally high urea content was encountered in four instances only, Cases 7, 27, 38, and 43, in all of which the phthalein was low. In Cases 38 and 43, urea was a better index to the renal condition than the cryoscopy, which, as has been pointed out, gave normal values. It would therefore appear that the urea may afford indications of cumulative phenomena earlier than the cryoscopy.

In no instance was a normal blood urea associated with markedly decreased phthalein. Case 37, however, with 27 per cent phthalein for 2 hours showed 0.35 gms. urea per L., while the δ was −0.61.

From a consideration of these data it appears that our previous claims in relation to the value of phthalein were well founded. The phthalein undoubtedly affords the truest index of the functional capacity of the kidney in this group of cases. In no instance was its findings incompatible with the clinical condition and subsequent history. Corroborative evidence only was adduced from the employment of the other tests, particularly valuable in this connection being the presence of cumulative phenomena. In several instances, however, the tests of retention failed to indicate the serious nature of the renal change, so that the findings of these tests can only be considered of great value when marked retention is indicated. The almost constant suppression of lactose in these cases, although rendering the test useless, is an interesting phenomena. From the standpoint of explanation, this is worthy of investigation. Urinary diastase and urea are of about equal value and are unreliable indices to renal functional capacity generally, but where very low values are persistently indicated they have some prognostic significance.

Cumulative phenomena do not occur in the presence of good renal functional capacity, and consequently the tests are unnecessary where the phthalein is normal. They do not occur constantly or early in presence of even marked renal involvement, and consequently would prove misleading in a certain proportion of cases with phthalein not employed. Positive cumulative phenomena, however, are of the greatest prognostic significance.

CONCLUSIONS

To detect the functional impairment associated with the renal changes consequent upon obstruction in the lower urinary tract, the phthalein affords an indispensable index to the true renal condition. Nothing beyond corroborative evidence is to be obtained from the employment of other functional tests.

THE INTERCAROTID PARAGANGLION AND ITS TUMORS

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SURGERY

By Donald C. Balfour, M. D.

TUMORS of the intercarotid paraganglion have, as a rule, been found malignant and recurrences following their removal are frequent. Whether this be due to the fact that the tumors are seldom operated on in an early stage or because of a high degree of primary malignancy is not clear. In the cases heretofore reported the tumors have varied considerably in size and frequently have been designated as perithelioma in type. The diagnosis is rarely, if ever, made before operation and usually not until pathologic examination reveals the histologic picture.

The removal of a tumor of this gland, because of its situation and malignancy, often entails somewhat drastic measures. Its position in the carotid notch in certain cases necessitates ligation of one or all of the main arterial trunks. In the case reported herewith removal of 4 cm. of the internal jugular vein was necessary.

The patient operated on in the Mayo Clinic was a woman 34 years of age who came for examination because of the distress incident to a large degenerating adenoma of the thyroid. The history of the patient was not suggestive except in regard to the gradually increasing pressure and severe pain radiating through the right shoulder and down the right arm. The physical examination revealed an enlargement of the thyroid 5 x 6½ inches, marked evidences of obstruction in the deviated trachea, and dysphonia. Laryngoscopic examination demonstrated a paralysis of the right vocal cord. Otherwise the physical findings were inconsequential.

On April 5, 1913, under light ether anæsthesia, the right lobe of the thyroid, which contained the degenerating adenoma, was removed. After completing the removal of the gland and ligating the vessels, further investigation revealed a separate mass in the right submaxillary region about the size of a lemon, having the appearance of a degenerating fœtal adenoma. At first this was believed to be an aberrant thyroid, but on attempting its removal it was discovered to be of an entirely different character, suggesting the probability of some malignant condition. The tumor was firmly adherent to all the surrounding structures and apparently had been crowded up into the submaxillary triangle by the enlarged thyroid. After freeing it in front and below it was found on deep dissection to be intimately connected with the deep vessels. The mass was dissected from the carotid vessels, but it had invaded the internal jugular vein and it was necessary to remove about 4 inches of this structure. This invasion of the vein made certain the malignancy of the tumor, and although the growth was removed *en masse* it seemed probable that more or less rapid recurrence would take place.[1] The field of operation was swabbed with Harrington's solution and a 24-hour pack of the same used to inhibit implantation of malignant cells. The patient's convalescence in the hospital was uninterrupted; she left on the sixth day. During her stay in town she suddenly developed a hemiplegia, which persisted until she left for her home. A recent report indicates the persistence of the hemiplegia and a probable local recurrence.

The after-history of patients in whom tumor of the intercarotid paraganglion has been removed is not encouraging. A cure without permanent disability is rare. Keen (1), who first called the attention of the American profession to the condition, states that only 7 patients of the 27 cases collected by him (including one of his own) recovered without complications. Ordinarily, if the patient does not succumb to recurrence, some disability which is usually due to brain-lesion occurs.

ANATOMY, PHYSIOLOGY, AND PATHOLOGY

By Franz Wildner, M. D.

Anatomy. In 1743 Albrecht von Haller (2) found a nodule about the size and shape of a kernel of wheat at the point of bifurcation of the common carotid artery. Anatomic dissection revealed that the little body was inserted in the fibers of the sympathetic nerve plexus which forms a dense meshwork around the common carotid artery and its branches; it may be found either at the crotch of the artery or in its immediate neighborhood closely adhering to the wall of the vessel. The common carotid artery or one of its branches gives off a small vessel supplying the arterial blood.

[1] Further description of the tumor may be found in Dr. Wildner's article.

Von Haller (2) based his conclusions on the findings obtained by gross examination and regarded it as one of the numerous ganglia distributed into the course of the sympathetic nervous system. He named it "intercarotid ganglion," thus classifying it as a nerve-structure.

In 1862 Luschka (3) made several hundred examinations with the object of studying its anatomy. The carotid body was found in every case. This author was the first to make detailed microscopic examinations of the tiny organ.

According to his findings, the carotid body is a globular or oval-shaped structure measuring from 5 to 7 mm. in length, 2½ to 4 mm. in breadth and 1½ mm. in thickness. It is found either in the bifurcation of the common carotid artery or on the posterior wall of the internal carotid, so that it is not distinguishable anteriorly. For this reason it has been termed retrocarotid body by French writers.

Its color is a greyish red up to a shade of purple-red, depending on the amount of blood filling the capillaries. It is fairly dense, and occasionally it may be broken up into halves with more or less of a connecting link between, rarely in four or five particles which are scattered over quite an extended area and in the course of the internal carotid artery. In this respect it resembles the parathyroids, of which especially the upper one is sometimes divided into two or more units, with occasional cell-groups in the lateral lobes of the thyroid completely surrounded by thyroid acini. A homologous organ is present in all mammalian animals at the bifurcation of the carotid or in its vicinity.

On microscopic examination Luschka (3) found cells with a large protoplasmatic body grouped into clusters and strands of various size, a large number of thin capillaries surrounding the cell-clusters, and very numerous sympathetic nerve-fibers with scant ganglion cells. The parenchymatous cells were the characterizing feature and, assuming their epithelial origin, he presumed that the anlage of the carotid gland buds off from the epithelium of a branchial cleft. He regarded the enigmatic body as a gland closely attached to the cervical portion of the sympathetic nervous system, and applied to it the term "intercarotid gland." He placed it in series with the adrenals, the anterior or glandular lobe of the hypophysis, and the coccygeal gland.

The carotid gland, the anterior lobe of the hypophysis, and the coccygeal gland, though differing in minor details to the extent of establishing their individual independence, are similarly constructed: Large cells resembling epithelial cells arranged into lobules of various size and shape in close anatomic relation with the sympathetic nervous system. The coccygeal gland found just in front of the apex of the coccygeal bone and associated with the median sacral artery is about 2 to 3 mm. in diameter and bears closer resemblance to the carotid than to any of the other glands. It is a constant single organ. Not infrequently it is represented by five or six small nodules. It contains a great number of capillaries and many fibers from the ganglion impar of the sympathetic.

These organs belong to ductless glands, blood-vessel glands or glands with internal secretion, following Claude Bernard (4), who distinguished the external secretion of bile — through an efferent duct — from the storage and mobilization of glycogen as internal secretion.

It is interesting to note that thirty years before the first cases were reported by Marchand (5) and Paltauf (6), Luschka (3) expressed the opinion that the vestige of the carotid gland may give rise to the growth of tumors.

Arnold (7) took a view different from that of Luschka (3). He was impressed by the large number of capillaries which occasionally formed whorl-like structures comparable to glomeruli, and concluded that the organ was characterized by a network of capillaries, the walls of which were coated by large cells. To him it was a vascular structure which he named "intercarotid arterial glomeruli."

Waldeyer (8) accepted Arnold's (7) view in the main. He regarded the large epithelial-like cells as especially differentiated cells of the adventitia of the capillaries and therefore as constituents of the vessel-wall. He made a distinction between the endothelium of the capillaries and the large perithelial cells and introduced the term "perithelial

organ" for organs built on the plan of the carotid or coccygeal gland.

A new era has been opened by Schaper's (9) article on the histology of the carotid gland. He showed that many microscopic findings described as the carotid gland were artefacts due to autolysis of the very unstable protoplasm of the cells. He emphasized the examination of fresh tissue and demonstrated that only chromic acid or its salts were able to fix the cell structure in a way reflecting the picture of fresh tissue. He pointed out that the epithelial-like cell-groups, notwithstanding their close relation to the capillaries, are independent of them, the capillaries showing a continuous endothelial lining and the same structure as elsewhere in the body.

Stilling (10) agrees with Schaper (9) as to the independence of the cells from the capillaries, and he finds that some of the cells take a brown tinge after fixation with bichromate of potassium; the self-same cells are found in small corpuscles attached to the abdominal part of the sympathetic, which he is inclined to class as accessory adrenals. In summarizing he thus emphasizes the structural analogy of the carotid gland with the adrenals: "I believe myself to be able to confirm that the carotid gland, whatever be its embryonic origin, is neither a simple vascular network nor a rudimentary organ, but a 'blood-vessel gland' of a structure analogous to that of the adrenal."

In a very logical investigation, Alfred Kohn (11) showed why so many authors, though unraveling numerous elements of truth, failed to arrive at a satisfactory explanation. They had studied the fully developed organ and had allowed themselves to be carried away by some dominant features on which they laid undue stress. They had in turn regarded the minute structure as a ganglion, a gland, an epithelial organ, a vascular organ, and a rudimentary organ. Kohn took the position that the nature of an organ is determined by the nature of its specific cells. His was an histogenetic viewpoint, and he attempted to trace the development and the textural structure from the earliest stage up to the developed organ. First of all he disproved the alleged epithelial character of the cells. Epithelial cells are found lining outer or inner surfaces of the body; they show a clean-cut borderline toward the underlying connective tissue and a polar orientation, the free surface being differentiated from the base. The carotid cells are found in the midst of connective tissue and heaped up without any trace of orientation or polarity. He concludes that the utmost lack of regularity as to size and arrangement of the cells rules out their epithelial nature, and contends that they are a type of cell altogether different from epithelial, endothelial, or perithelial cells. Another peculiar feature of the cells is the unstability of their protoplasm. Specimens removed later than 45 minutes after death showed marked changes of the cells due to autolysis of the cell-substance. Experimentation with the various fixing fluids proved that only chrome salt solutions are likely to fix and preserve the protoplasm in its natural condition, at the same time staining it often a faint yellowish tint or any shade from a light yellow to dark brown. All other fixatives, as formalin, absolute alcohol, or sublimate, dissolve certain substances of the cell-body, the nuclei only remaining intact in a protoplasmic network with irregular meshes. Kohn named the cells "chromaffin cells" on account of their chemical affinity to chrome salt solutions. These cells are large and mostly polyhedric, though very polymorphous in appearance; the protoplasm is almost homogeneous or faintly granular. The nucleus is round or oval and shows a dense chromatin network with one or more nuclei and a sharply outlined nuclear membrane. Mitoses are found, though rarely.

A very large number of non-medullated nerve-fibers enter into the formation of the carotid gland; typical ganglion cells are found scattered or in small groups in the sympathetic nerve fibers. Kohn (11) then proved by comparative studies the close relation between the cells of the carotid gland — the chromaffin cell — and the non-medullated nerve-fibers. He found groups of chromaffin cells embedded in the sympathetic nerve-fibers (Fig. 1) running to the carotidgland much as ganglion cells are interwoven into the tracks of nerves. In the supreme cervical ganglion of the sympathetic

small groups of chromaffin cells are found. There is a continuous stretch of chromaffin cells extending from the carotid gland through sympathetic nerve-fibers to the supreme ganglion of the sympathetic. Here typical ganglion cells, chromaffin cells and nerve-fibers are found in closest union. The finding of the chromaffin cell — the characteristic element of the intercarotid gland — in sympathetic nerve-fibers and ganglia led to the assumption of a closely allied kinship between the carotid gland and the sympathetic nervous system.

Small islets of chromaffin cells are found in the sympathetic nerves and ganglia from head to coccyx (Fig. 2). Large autonomous organs are built up in the retroperitoneal space in close neighborhood to the abdominal aorta. The most proximal part of the abdominal paraganglion dips down into portions of the Wolffian body and the union of these two widely different organ-systems shapes the adrenal gland (Fig. 3). Its medullary part is a part of the sympathetic derived from the ectoderm, while the cortical part is part of the renal system derived from the mesoderm.

Another large paraganglion is the "accessory organ of the sympathetic" (Zuckerkandl (12)) found at the site of bifurcation of the abdominal aorta, which is well developed in the new-born, being 10–15 mm. in length and 3–5 mm. in width. Small paraganglia are found in the space betweeen the adrenals, kidneys and ureters, in the interiliacal angle, in the mesorectum and broad ligament (11) (Fig. 4). All paraganglia outside the adrenal medulla undergo involution after birth.

The chromaffin cell, which is the structural unit of the carotid gland, the medulla of the adrenal, and the accessory adrenals of the sympathetic, is a derivative of embryonic cells of the sympathetic. The embryonic cells of the sympathetic — sympathoblasts — differentiate into adult sympathetic ganglion cells and nerve-fibers, but some of them form chromaffin cells, *a cell which retains to a certain extent the features of the embryonic ganglion cells*. The author believes that the undeniable epithelial appearance which so deeply impressed itself on many workers may be a heritage of the epithelial origin of the nervous system, which traces its derivation back to ectodermal epithelium. Thus the chromaffin cell is closely related to the ganglion cell, but it does not form fibrils which are characteristic of the ganglion cell. Like the ganglion cell it appears either singly or in smaller or larger groups embedded in the track of sympathetic nerves, or, like the more differentiated ganglion cell, it may form individual organs. Kohn (11) introduced the term paraganglion for organs made up of chromaffin cells to express their histogenesis from the embryonic ganglion cell.

Another term "parasympathetic organs," expresses their histogenetic and anatomic relationship. Chromaffin cells develop in all parts of the sympathetic nerve anlage, in the cervical as well as the abdominal and sacral part of the sympathetic system; yet in certain locations they form large aggregations and independent organs. The intercarotid paraganglion, the adrenal paraganglion — the largest of the body — and the abdominal paraganglion are all made up of chromaffin cells derived from the embryonic sympathetic cell. They all retain their initial kinship with the sympathetic system in the fully developed organism.

In the human new-born chromaffin tissue is very widely distributed, but an involution sets in in early life and no chromaffin cell is found in the adult sympathetic (rests of chromaffin cells remaining in the carotid gland and the adrenal). In mammals it is a permanent structure (Vincent).

It has been shown in embryos of the cat, pig, and man that the cells of the intercarotid paraganglion develop from embryonic ganglion cells of the sympathetic at the ventral side of the internal carotid artery (Fig. 5). While the majority of the embryonic sympathetic cells develop into typical sympathetic ganglion cells and nerve-fibrils, a small number form sphere-shaped groups, the beginning of the alveolar structure of the zellballen (Fig. 6). These sharply defined zellballen are the unit of the adult intercarotid paraganglion; they take up at a later stage the chromaffin reaction. The fully developed organ then recedes from the wall of the internal carotid artery and descends to a point at or near the bifurcation;

on its way it has dragged sympathetic nerve-fibers with typical ganglion cells along, leaving in its trail a few scattered chromaffin cells. Kohn's (11) view of the nervous origin of the intercarotid paraganglion has been confirmed by subsequent investigations. It is interesting to consider that we have returned to the position taken by von Haller (2) in 1743. He based his sound and simple argument on the facts disclosed by gross dissection; slightly modified, his standpoint represents the best supported modern views.

In summarizing, one may say that the chromaffin cell — the structural unit of the intercarotid paraganglion — is found in the whole realm of the sympathetic nervous system, proving its histogenic unity. Although the chromaffin cells are derived from the sympathetic nervous system, yet there is good evidence that they perform an internal secretion in adult life. The internal secretion of the adrenal has been established since the time of Addison (13) in 1855. It is reasonable to use the term gland for secreting organs no matter what their morphologic origin is. Apparently the terms paraganglion and gland will be used promiscuously for the aforesaid organs, depending on the point from which they are viewed. There seems to be a close union between the nerve and vascular system in the paraganglia, the chromaffin cells throwing their secretion directly into the circulation under the stimulation of the sympathetic nervous system.

Physiology. The intercarotid paraganglion shows, like all other paraganglia, an extremely rich vascularization. This fact in itself seems to indicate a very active metabolism. Only the largest paraganglion of the body, the medulla of the adrenal, has thus far received due attention. Adrenalin, when administered intravenously, will cause marked increase of arterial blood pressure, due to its contracting effect on the unstriped muscle-fibers of the arterial wall, combined with decrease in pulse rate and increase of the force of the heart beat. Glycosuria also results. Claude Bernard (4) produced glycosuria by stimulation of a point at the floor of the fourth ventricle and concluded that the stimuli traveled by way of the sympathetic system to the liver, causing a glycogenolysis. In recent studies on rabbits it has been shown that this experiment fails after removal of the adrenals or severance of the sympathetic or splanchnic nerve. In the light of this additional knowledge it seems that stimulation of certain points of the brain may be conveyed in the track of the sympathetic to the adrenal medulla, causing a secretion of adrenalin with its glycosuric effect. Adrenals removed from animals on which Bernard's (4) experiments had been made showed a lesser pressor effect on the vascular system than the intact adrenal; and microscopic examination disclosed diminution or absence of the chromaffin reaction of the cells of the medulla. The various stages of physiologic activity of the secreting cells correspond to definite morphologic pictures, and the secretion appears in the shape of droplets which are evidently thrown into the thin-walled capillaries.

Josue (14) found in rabbits which he had injected for some time intravenously with three drops of adrenalin 1:1000, arteriosclerosis of the aorta and larger arteries.

There are some observations that seem to lead to the assumption of an important rôle played by the intercarotid gland. The wealth of sympathetic nerves running into it, the chromaffin cells in immediate contact with capillaries, only a thin endothelium intervening, the unstability of its protoplasm, and its rapid autolysis after death seem to indicate the presence of a highly sensitive chemical substance.

The writer was able to find only a single instance of an experimental investigation into the function of the carotid paraganglion. Frugoni (15) cites Vassale (16) who noted, after bilateral destruction of the organ, a considerable glycosuria of a passing nature or cachexia leading to death. Frugoni (15) showed that intravenous injection of 5 ccm. press juice obtained from the carotid glands of suckling calves would kill a rabbit in one or a few minutes; there were convulsions, paralysis of respiration, and failure of heart action. The blood of the injected animal lost its clotting property and the visceral organs were markedly hyperæmic. The main action seems to be on the vascular system. Small doses of

extract have a depressor effect on the vascular system; it is antagonistic to adrenalin.

Pathology. Marchand (5) published the first case of a tumor of the intercarotid paraganglion in 1891. Paltauf (6) reported four cases in 1892. The most striking feature of all these tumors was their location at the angle of bifurcation of the common carotid and their close adherence to the wall of the arterial vessels. In four out of these five reported cases the tumors could be removed only with resection of the common carotid artery and its branches, while in the fifth case the tumor was shelled out without damage to the vessels. The tumors showed also a great similarity in their microscopic structure. Marchand (5) concluded that the tumor was of a peculiar structure, which could be derived only from a certain well-defined anlage. Paltauf (6) regarded the four tumors he reported as peritheliomas of the carotid gland. The conclusions arrived at by these two authors are the more convincing as they were drawn independently. Since then, about 35 cases of tumor of the intercarotid gland have been reported in the literature. With three exceptions they were all removed by operation. The three tumors found at post-mortem are among the smallest in the series, being about the size of an almond or English walnut. The typical location of the tumors is the angle of the bifurcation of the common carotid, the growth as a rule adhering to the wall of the artery. Sometimes the common carotid artery is seen passing into the tumor (Scudder (17)). In other cases the arteries lie in deep grooves in the tumor.

When the tumor is firmly adherent to the wall of the vessel its removal is feasible only after resection of the common carotid artery and its branches. In some cases the internal jugular vein, the sympathetic, vagus, hypoglossal and lingual nerve have been removed together with the carotid tumor. In a few cases the tumor, being loosely attached to the wall of the vessel, could be shelled out without damage to the arteries, or with ligation of the external carotid only (6, 18, 19, 17, 20).

The size of the tumor, which is well encapsulated, may be that of an almond, usually that of a hen's egg, and in some cases of a goose egg. Some have the shape of a potato and have been termed "potato tumors" of the neck.

The color depends on the color of the tissue proper and the amount of blood filling the capillaries. It may be a white, reddish grey, brownish red or even dark purple. The transparency and the glossy appearance of a freshly cut surface have been noted in several cases, occasionally reminding one of a parenchymatous goiter (1). The consistency depends on the relative amount of cells and fibrillary tissue; it may be firm, hard, rather elastic, or sometimes as soft as brain tissue. The capsule is fibrous, smooth, thin and contains a large number of thin-walled veins. Fine strands of connective tissue run from the capsule toward the center, producing a lobulated appearance, and a sponge-like structure is produced by the presence of very numerous enlarged capillaries. The cut surface is characterized by its homogeneity and evenness of texture. In several cases that came to post-mortem a search was made for the intercarotid paraganglion at the bifurcation of the vessel and in the surrounding tissue, but no trace of it could be found, while it was invariably present and of normal size on the other side. It was further observed that the tumor derived its arterial blood supply from the common carotid or from one of its branches near the bifurcation in the same manner as does the intercarotid gland. The stroma of the tumors is usually more developed in the center than on the periphery.

On going over the tumors of the intercarotid gland one is impressed by the almost perfect identity of the salient features of all of them. There are certain differences in the various individual tumors and there are variations in the pictures of sections taken from various places, but these differences appear secondary and are due to degenerative changes that have taken place.

Microscopically the tumors show an alveolar structure with thin capillaries forming the stroma. The cells of the alveoli are grouped irregularly and show a homogeneous protoplasm without partitions between the individual cells. In other cases the cells are rather well defined, polyhedric or round. The

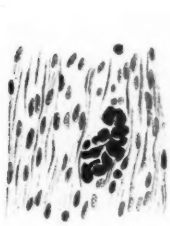

Fig. 1.

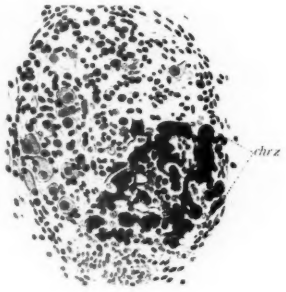

Fig. 2.

Fig. 1. Sympathetic nerve from the vicinity of the carotid paraganglion. In the midst of it a group of chromaffin cells.

Fig. 2. Sympathetic ganglion from the pelvic portion of the ganglion cord (fœtus of 7 months). One-half of the ganglion taken up by the chromaffin cells (chrz). (From A. Kohn.)

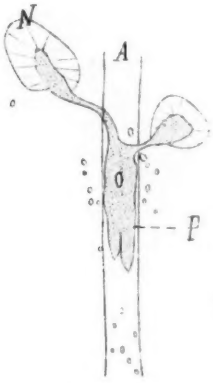

Fig. 3.

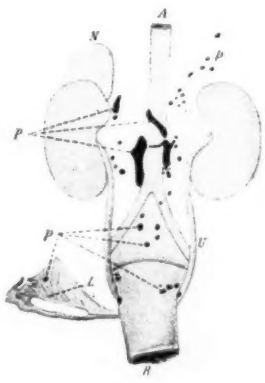

Fig. 4.

Fig. 3. Paraganglion of a new-born rabbit. Diagrammatic drawing, 10 X. A, aorta; N. adrenal; P, aortic paraganglion forms the medulla of the adrenals with its cranial terminals. (From A. Kohn).

Fig. 4. Paraganglia of a girl (45 days old). A, aorta; N, adrenal; U, ureter; R, rectum; L, broad ligament; P,p, major and minor paraganglia. On the left the adrenal has been removed in order to show the paraganglia on its posterior surface.

nuclei are round or oval, sharply outlined, showing a number of chromatin nodules immediately below the vesicular membrane, and resembling plasma cells. Mönckeberg (21) noted in two of his cases that the protoplasm of specimens fixed in Müller's or Zenker's fluid showed excellent preservation, while another tumor fixed in formalin showed the same foam-like structure of its cells as is shown by the formalin-fixed cell of the normal organ. He laid stress on this reaction as being the chemical proof of the origin of the tumor from the carotid gland. A few of the cells of one of his tumors took up the yellowish tinge characteristic of the chromaffin cell.

The capillaries show a continuous endothelial lining, the cells being set directly on the endothelium. The width of some capillaries is remarkable and accounts for the porous structure of some of the tumors. Hyaline degeneration is apt to set in in the walls of the capillaries, especially in the center of the growth.

Syncytial cell groups and giant cells are occasionally observed. Karyokinetic figures are seen in the tumors, sometimes in large numbers.

Tumors of the intercarotid paraganglion are made up of chromaffin cells, the elements which form the normal structure. Whenever a tumor of a paraganglion — a paraganglioma — is suspected, it should be fixed in a solution containing chromic acid or its salts. A mixture of ten volumes of 3½ per cent solution of bichromate of potassium and one volume of formalin gives an excellent fixative. After 24 hours' fixation the tissue should be mordanted in the bichromate solution for two days, then treated in the usual manner. The intercarotid gland may be divided into several portions and chromaffin cells found in places distant from the bifurcation. We may then find chromaffin tumors at a distance from the bifurcation, for instance at the jaw (22).

The tumors of the carotid paraganglion are rather benign, slow-growing neoplasms. They begin as hypertrophies of the normal organ and grow 5, 10, even 37 years (19) without causing any discomfort. Though they adhere very closely to the arteries, only one case hitherto reported (Marchand's (5)) showed penetration of the arterial wall and two small nodules of tumor tissue in the lumen, one exactly at the bifurcation and another a little higher up in the external carotid.

In the case herein reported, the tumor was removed from the right side of the neck of a woman 34 years of age. After multiple adenomas of the thyroid had been removed a mass

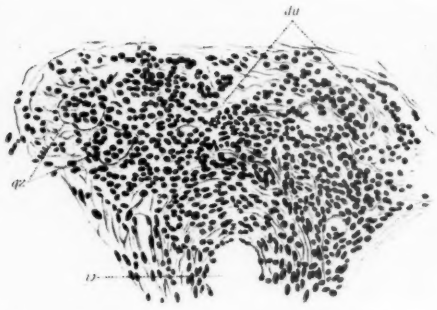

Fig. 5. Pig embryo, 45 mm. Section through nerve-plexus (n.p.) at ventral side of internal carotid (c.i.). Ganglion cells in diffuse arrangement. Grouping of cells into acini (z.b.) the anlage of the carotid paraganglion. (From A. Kohn).

Fig. 6. Pig embryo, 87 mm. The cell alveoli (z.b.) have increased in size and multiplied. The organ shows the finished appearance. (From A. Kohn).

was found lying high up in the neck running up to the angle of the jaw, very densely adherent to all surrounding structures involving the internal jugular vein, a portion of which was removed with the tumor. The tumor measured 5 x 7 x 4 cm., was well encapsulated (Fig. 7), and could be shelled out without injury to the arteries. It was of a greyish red color, very even on section, of a "live rubber" consistency and remarkable transparency (Fig. 8). The internal jugular vein was loosely plugged with tumor tissue, 4 cm. in length. The microscopic structure is analogous to the previously reported cases (Figs. 9 and 10).

Metastases in lymph-nodes have been noted in a few cases. Recurrences have been noted in about one-fourth the cases. Hyalin degeneration of the capillaries has been noted in a good many cases.

Systemization. The study of the organogenesis of the intercarotid paraganglion has shown its origin from the primitive sympathetic anlage and has attached this apparently isolated and vestigial corpuscle to the sympathetic nervous system. The embryonic sympathetic cell — sympathoblast or sympathogonia — develops into the sympathetic ganglion cell and nerve-fiber on the one hand and the chromaffin cell on the other.

Sympathoblast (Sympathogonia, embryonic sym. cells)

Sympathetic ganglion cell and nerve-fibers. | Chromaffin cell (Paraganglion, parasympathetic organ).

It is interesting to examine the neoplasms of the sympathetic in the light of the facts put forth. Elements representing the various stages of embryonic development and differentiation may go into the making of sympathetic tumors.

Sympathoblastoma (Lympathogonoma, ganglioma, embryonale sympathicum)

Ganglioneuro (fibro) ma. | Chromaffin tumor (Paraganglioma Parasympathoma).

Tumors consisting of these elements may occur at any point of the central nervous system—on the cerebrospinal nerves, on the sympathetic with its distribution, and in the visceral organs.

The sympathoblastoma may occur anywhere within the boundary of the sympathetic, preferably in the adrenal medulla of newborns or children. It is a malignant tumor with metastasis to the liver and other organs (23, 24, 25, 26, 27, 28, 29, 30). Full-blown ganglion cells and nerve-fibrils constitute the ganglio (neuro) fibroma. They may arise from the ganglionated cord (31, 32, 33); they may be interposed between kidney and adrenal (34), or between spine and kidney (35). Wegelin (36) found 14 out of 18 cases of the ganglionated cord on the left side. Herxheimer (37) found among 28 tumors 17 arising from cord ganglia, 11 from the medulla of the adrenal or its vicinity.

A chromaffin tumor — a paraganglioma — may develop from any paraganglion of the body; so far only few tumors have been ascer-

Fig. 7. Case No. 82318. Outer surface of tumor with resected internal jugular vein at upper pole plugged by tumor-tissue.

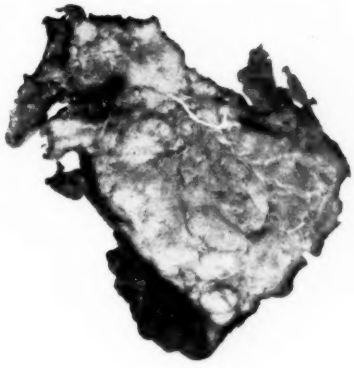

Fig. 8. Case No. 82318. Cross section of same tumor.

tained (38, 39, 21, 40, 41, 42, 43). These cases are tumors of the adrenal and intercarotid paraganglion and the coccygeal gland.

The adrenal medulla may develop all the possible tumors of the sympathetic tissue; the embryonic neuroblastoma, the ganglioneuroma and the chrome-brown tumor or paraganglioma. The cortex of the adrenal produces the so-called adrenal hypernephroma as typical tumor (44, 45).

Sacrococcygeal tumors should be carefully examined for the presence of chromaffin cells; they may be derived from the coccygeal gland (40, 46). Tumors of the nervous system should be fixed in a formalin bichromate mixture in order to preserve the chromaffin cells.

Excessive and defective physiologic activity associated with tumors of the glands of internal secretion. What is the relation between the neoplastic cell and its normal mother-cell as to physiologic function? From microscopical examination, production of bile by the cells of a carcinoma of the liver and production of colloid in carcinoma of the thyroid have been known for a long time. The metastases of these tumors may occasionally secrete bile or colloid. Even under such conditions as exist in the bone-metastasis of a thyroid carcinoma, colloid in the acini has been seen. On the other hand, we have clinical evidence that the carcinoma of the thyroid may aggravate the symptoms of hyperthyroidism. A degenerating fœtal or adult adenoma may produce toxic symptoms without exophthalmos (45).

Assuming a correlation between the changes in form and the changes in function, we may expect a hyperplasia of an organ to be combined with a hyperfunction, perhaps followed later on by hypofunction with the beginning of secondary tissue degeneration. Acromegaly is associated with an adenoma of the eosinophilic type of the anterior lobe of the hypophysis (47, 48).

Tumors arising from the posterior lobe of the hypophysis may bring about the clinical symptoms of a high limit of sugar assimilation, adiposity, lowered blood pressure, slowed pulse, subnormal temperature, asthenia, and drowsiness (49).

Neoplasms of the pineal gland of young males may bring on rapid growth of stature, hirsuties and a premature sexual development, with corresponding change of the frame of mind (50).

Of the tumors of the adrenal we have to differentiate between those arising from the cortex and those from the medulla. The adrenal hypernephroma is apt to produce abnormal sex characteristics when present in females. The symptoms seem to be the more pronounced the younger the individual. Hirsuties, especially on upper lip, neck and chin, and chest, cessation of menses, atrophy of uterus and ovaries, obesity, and a change of voice have been observed (51); in men, a

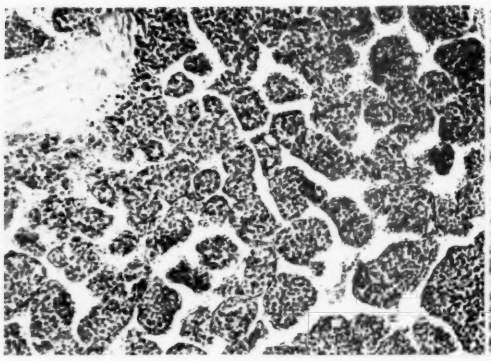

Fig. 9. Case No. 82318. Microscopic section, low power, showing the capillaries and alveolar cell-groups.

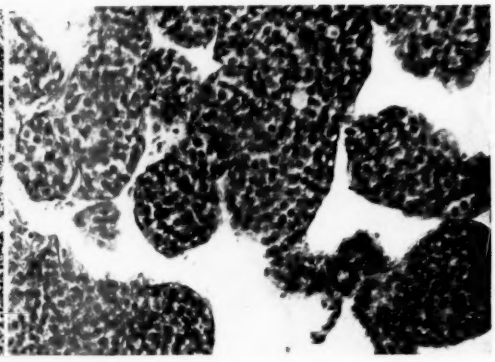

Fig. 10. Case No. 82318. Same section, high power, capillaries with a continuous endothelial lining. Note the nuclei of endothelial cells.

gigantic increase in bodily strength has been observed (45).

Chromaffin neoplasms of the adrenal medulla, with the symptoms of hyperfunction of the adrenalin-producing tissue, were observed in a few instances. The clinical symptoms were those that can be experimentally produced by repeated adrenalin injections: glycosuria, hypertrophy of the heart, especially of the left ventricle, high blood pressure, arteriosclerosis (found in a baby 2 years of age) (52).

In view of these observations on the evidence of physiologic function in the cells of neoplasms of glands of internal secretion, one would expect to find evidence of the physiologic function in the presence of the enormous overgrowths of the intercarotid gland. In none of the reported cases of tumor of the intercarotid gland has a hyperfunction of chromaffin tissue been noted; perhaps one of the reasons may be that it has never been looked for.

Function, pathology and clinical manifestations of the chromaffin system, the intercarotid gland in particular, are little known, and the cases of tumor of the carotid gland have usually been diagnosed at the operating table, after having been designated as lymphoma or adenoma of the thyroid. The tumors of the intercarotid glands are by no means as rare as has been generally supposed. The Index Medicus shows three papers for the first six months of 1913.

It would seem rational that in cases of tumors of the carotid gland the whole chromaffin system be examined and evidence of its hyperfunction be carefully sought after. The writer would tentatively advance the hypothesis that the carotid gland tumor is but an anatomic expression of a functional disorder of the chromaffin system, affecting one or more units of the same. A bilateral overgrowth of the carotid gland has been observed by Enderlen (53).

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CONCERNING APPARENT CURES OF RENAL TUBERCULOSIS

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AMONG the many interesting features of renal tuberculosis none is more important than the apparent cures of the disease. The dispute between the relatively few practitioners who would treat renal tuberculosis without operation and the many who urge prompt nephrectomy, as soon as the diagnosis is made, can only be settled by a clearer understanding of what the apparent cure of renal tuberculosis means. The underlying difficulty in reconciling the opposing views is probably a lack of thorough understanding of the relation between the pathological lesion in the kidney caused by tuberculosis and the symptoms arising therefrom. Thus it is not generally appreciated that the variations in the patient's general health as well as in his renal or vesical symptoms are due to alterations in retention (renal or vesical), or to changes in the activity of pelvic or vesical lesions, rather than to changes in the kidney parenchyma.

Every case of early renal tuberculosis submitted to operation places in our hands a pathological specimen suggestive of the fact that the renal lesions are far older than the duration of symptoms would suggest. Indeed, the mere presence of surgical tuberculosis in the parenchyma of the kidney causes no symptom. Upon this fact it is scarcely necessary to insist. The familiar case history with its exclusively vesical symptoms is sufficient evidence that the lesion in the kidney parenchyma does not make itself felt until one of two things happens. Either it extends through the capsule and excites perinephritis, or else it extends to the pelvis and there sets up a tubercular pyelitis.

Perinephritis is rare, and need not concern us.

Tubercular pyelitis is common. Doubtless the painful and frequent urination, which is so often the most prominent early symptom of the disease, is at the outset due entirely to tuberculosis of the pelvis and ureter. Judging from the specimens I have seen, this tuberculosis usually takes the form of a disseminated, subepithelial infiltration of the mucous membrane with little tubercles which give to the mucosa a pebbled appearance, similar to that produced by pyelitis cystica.

At this early stage, the cavity of the kidney pelvis is usually very slightly dilated (apparently by relaxation of its walls). There may be no ulceration excepting that upon the papillæ, the source of pelvic infection. Yet even with lesions so slight as these, the patient may be urinating every hour, with considerable straining and terminal pain. If the kidney is removed at this time, the frequent urination is checked as if by magic and does not return.

The next important pathological change consists in the extension of the tuberculosis to all the layers of the pelvis and ureters and to the bladder. Upon the mucous surface the tubercles ulcerate, and upon the outside they excite that adhesive inflammation about the pelvis and ureter, with irregular thickening of the walls of these channels, which is so characteristic of renal tuberculosis. As a result of this ulceration and infiltration, the lumen of the excretory channel becomes more or less completely obstructed and the region above the obstruction dilates. Inasmuch as the cavity of the kidney pelvis itself often becomes almost completely obliterated by this process, the dilatation may affect only one portion of the kidney, though it usually affects the whole kidney. Exceptionally the obstruction is in the ureter and the pelvis itself is widely dilated instead of being contracted.

As a result of these pathological changes, renal colic may occur from the passage of blood clots or shreds of fibrin or tissue, or pain in the loin may result from distention of the kidney, or perinephritis; while a more or less definite sensitiveness and dragging pain in the side may result either from the tension in the kidney and its pelvis, or from the dragging of the shortened and thickened ureter, or from perinephritis.

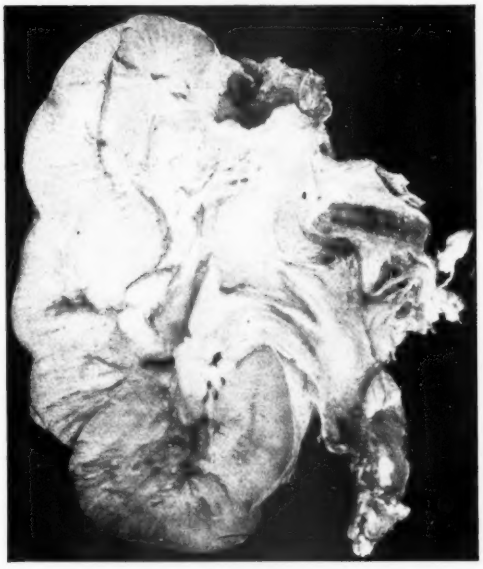


Fig. 1. Kidney sclerotic, pelvis a little thick. Ulcerated papilla at lower pole. The caseous upper pole has been torn away.

Fig. 2. Leaded catheters in both ureters. Collargol injection of right kidney (left in figure) shows dilated calyces and suggests enlargement of lower pole.

But if the ureter is not shortened, and if the retention either in one pole of the kidney, or the whole kidney, is sudden and complete instead of being gradual and incomplete, the patient's discomfort may suddenly cease (either with or without renal colic). Even though the tuberculosis has extended down the ureter beyond the point of stoppage, if urine ceases to flow through the ureter the symptoms will promptly cease, whether the flow of urine be checked by nephrectomy or by plugging of the duct.[1]

In the light of these facts, the varying history of patients with renal tuberculosis is readily understood. The great majority of them constantly have frequent and painful urination, and though from time to time very considerable remission of the symptoms may occur, these never entirely disappear, and as the inflammation progressively invades more and more of the bladder the general tendency is for the urinary distress to become more and more marked. Some patients fail rapidly in general health on account of partial retention, usually in the kidney pelvis, less often in the bladder. But the exceptional patient may continue to have fairly good drainage and exhibit the most extraordinarily good general health.

On the other hand, once in a long while, one encounters a case of renal tuberculosis whose symptoms have suddenly and completely ceased and who has apparently regained perfect health without operation. Such a case I have seen but once. The patient suffered in the early '90's from many renal colics upon the right side and from a certain amount of bladder irritation. Tubercle bacilli were found in the urine. In 1895 Dr. McBurney explored the right kidney but failed to find anything wrong with it and did not remove it. Within a few days of this operation, the patient had a frightful renal colic, and thereafter all of his symptoms ceased. He remained entirely well until 1912, when he again began to have pains in the right side and rapidly lost weight. At the time I saw him the right ureter seemed

[1] Indeed, it seems possible that partial obliteration of the kidney, resulting in stoppage of the infected flow, but with no stoppage of the *uninfected* urine, may equally be followed by cessation of symptoms.

Fig. 3. Parenchyma normal. Pelvis pebbled. Cystic lower pole due to healed tuberculosis.

absolutely occluded: it transmitted no urine and would admit no catheter, while the left kidney and bladder seemed entirely normal. The diagnosis of carcinoma of the gall-bladder and liver was made and confirmed by the prompt death of the patient. The right kidney had evidently ceased to functionate at the time of the last renal colic and had been a dead organ for 17 years, thereafter. With its shutting off the lesion lower down the urinary tract subsided.

That this is an unusual case is attested by the experience of every one of us. I have operated upon thirty-one cases of renal tuberculosis and seen perhaps as many more who have either been operated upon by other surgeons or who have refused operation, and never yet have I seen its mate. Several of my cases, however, have shown for long periods of time a very considerable remission of symptoms. Two of them have remained entirely without symptoms, respectively, for two and six years, at the end of which periods the symptoms returned and I removed the kidney.

The first of these patients, an Irishman, 27 years of age, came to St. Vincent's Hospital in February, 1911, giving the following history: Never had venereal disease; was a heavy drinker; had pneumonia two years before admittance and showed a scar on his neck from a gland which suppurated at that time. His urinary history was one of hæmorrhage; six years before he had had a profuse, spontaneous, painless hæmaturia. This ceased after a day or so. Three months before admission the bleeding reappeared and lasted two days. At the time of entering the hospital he had been bleeding again for seven days. He showed no other symptoms relative to his urinary organs and had lost no weight. As soon as he entered the hospital the bleeding stopped and he went through an attack of delirium tremens, lasting two weeks. At the end of this time physical examination revealed a very large, low kidney on the right side and a slight pulmonary dullness at the left base. In 24 hours he passed 47 oz. of urine containing 1.3 per cent urea, some red and white blood cells, and no pus. Phenolsulphonephthalein was injected and after a delay of 12 minutes he passed 20.5 per cent in the first hour and 8.9 per cent in the second.

Cystoscopy showed a normal bladder and normal ureter orifices. No. 6 flute tip ureter-catheters were readily introduced 25 ccm. into each ureter and in the first 8 minutes 5 ccm. of urine were obtained from the enlarged right kidney, containing, besides the usual blood and epithelial cells, numerous casts, a few leucocytes and 1.3 per cent urea; while from the left one only 3 ccm. were obtained with 0.3 per cent urea, no pus and no casts. The phenolsulphonephthalein appeared in 8 minutes from the right kidney; in 9 from the left. In the first 30 minutes, thereafter, 3 per cent was excreted from the right side, only a trace from the left and in the following 30 minutes 5.6 per cent from the right side and 1.7 per cent from the left. There was no pus in any of the urinary specimens.

Here was a curious state of affairs: The patient's symptoms suggested tumor, and the marked enlargement of the right kidney suggested that the tumor was unquestionably on the right side, yet the functional test showed a great deficiency in the excretion, both of urea and phenolsulphonephthalein from the left kidney. Accordingly I cut down, first upon the left kidney, found its upper third converted into a cheesy mass, obviously an ancient tuberculosis. Turning the patient over, a small incision sufficed to reveal that the right kidney was very low, hypertrophied to twice its normal size, but otherwise apparently normal. Turning back to the left side, I therefore removed the left kidney; the patient making an uneventful recovery. Maximum temperature after the operation was 100.4° F., and the wounds healed by primary union.

On looking back at the case, the presence of the casts in the urine from the right kidney and the markedly deficient excretion from the left side, together with the absence of casts, should have warned me of the presence of

renal tuberculosis on that side, had I but seen some pus. For casts on one side and deficient function with pus and without marked enlargement of the kidney upon the other side is very suggestive of unilateral tuberculosis.

But the second, and for our present purpose the most important, feature of this case is the distribution of lesions of the infected kidney (Fig. 1). Its upper pole was converted into a cheesy mass; the remainder of the kidney showed a few evidences of beginning tuberculosis scattered here and there, the most notable of which was an ulceration upon one of the lower papillæ. The pelvis of the kidney was not far from normal. We may make the pathological and clinical pictures coincide by assuming that the original lesions were at the upper pole and closed with caseation of that pole, that the kidney pelvis was but little infected, and that with spontaneous shutting off of the tuberculous lesion in the upper pole, the whole process became quiescent for six years, only to break out again with precisely the same process at the lower pole. If this patient had come to autopsy after being killed in an accident, at some time between the first and second outbreaks of his renal tuberculosis, his kidney might have gone down in history as proof that renal tuberculosis can and does heal with only partial destruction of the parenchyma. Fortunately, the science of medicine has been spared this shock.

My second case illustrates this temporary healing after partial destruction of the kidney even more strikingly. The patient, a woman 38 years of age, was brought by her physician for cystoscopy in July, 1910. She was in excellent general health and complained only of a spontaneous "cystitis" (i. e., frequent and painful urination with pus) of six months' duration, which had been entirely uninfluenced by treatment, but had ceased spontaneously a month before she came to me. A few days thereafter she had a single hæmaturia. Cystoscopy showed nothing abnormal in the bladder excepting a swollen, inflamed right ureter orifice. The ureters were both readily catheterized to the renal pelvis. Five ccm. of indigo-carmin was injected; this appeared from the left kidney in 13 minutes; from the right in 32 minutes. The urine from the left kidney contained 1.8 per cent urea, no pus and no casts; that from the right kidney contained 1.2 per cent urea, pus, no casts and no tubercular or other bacteria, by smear. Guinea pigs were inoculated with the bladder urine and survived.

She was seen again in November, 1910, and November, 1911, each time free from symptoms, but passing urine which contained a very faint trace of albumin and a few pus cells.

On January 31, 1913, she returned, stating that after a slight vesical irritability which had lasted for two months, she was suffering a distinct bladder irritation for two days. Cystoscopy showed a little vegetating mass at the right ureter orifice and some inflammation about this. Ureter catheterization drew from the right kidney urine containing 0.4 per cent urea, pus and tubercle bacilli by smear; while the urine from the left side showed 0.75 per cent urea and a few leucocytes. The X-ray showed an enlarged right kidney and an injection of collargol into this (Fig. 2) showed that while the pelvis was not dilated the enlargement was chiefly confined to the lower pole.

Upon these findings the right kidney was removed. Its lower pole was found replaced by two large cysts, containing bloody serum. The remainder of the kidney was so normal that a most competent pathologist was unable to find any lesions in it even suggestive of kidney tuberculosis (Fig. 3). But the pelvis, though normal in every other respect, was studded with little spots which proved on section to be miliary tubercles. Her symptoms ceased upon removal of the kidney; her wound healed by first intention.

Here, then, is a second case of partial healing of renal tuberculosis with remission of symptoms for two years. As we look at these two specimens, I think we may anticipate the day when specimens similar to these shall be made the basis of a plausible but entirely unwarrantable pathological attack upon nephrectomy for the cure of renal tuberculosis. To forestall this is my excuse for presenting them before you.

THE RÖNTGENOGRAPHIC DIAGNOSIS OF GALL-STONES AND CHOLECYSTITIS[1]

By LEWIS GREGORY COLE, M. D., New York City
Professor of Radiology, Cornell University Medical College

THE röntgenographic diagnosis of gall-stones and cholecystitis is a subject that has interested röntgenologists and diagnosticians since the earliest use of the X-rays. It seemed reasonable that the röntgenographic method of detecting renal calculi would likewise reveal biliary calculi. But repeated failures led most of us to abandon hope, and for a long time not even to attempt the examination. With the increasing number of cases examined, and the use of an intensifying screen and soft tube, gall-stones were occasionally detected. This led me to the routine examination for gall-stones in all cases presenting symptoms referable to the right hypochondrium. The number of positive diagnoses which I have been able to make proves that the examination is justified; but a negative diagnosis should never be made where there is no evidence of a calculus.

Several observers have reported individual cases where gall-stones have been detected. Three cases were reported by Pfahler in 1911.[2] In 1913 Thurston Holland reported three cases, and called attention to the necessity for differentiating right renal calculi from biliary calculi.[3] This point alone justifies careful consideration of the subject. Gall-stones and cholecystitis have been observed röntgenographically in 20 out of 500 cases examined for gastro-intestinal lesions, and I believe that the systematic search for them by the method described in this article will reveal them in a much larger percentage of the cases examined. Before studying the röntgenographic indications of gall-stones and gall-bladder infection, the anatomy of this region should be noted. There are three possible locations for stones, viz., in the gall-bladder, the cystic duct, and the common duct. But such considerations, and also the frequency with which infection occurs with or without calculi, are questions not within the scope of this communication.

INDIRECT EVIDENCE OF BILIARY CALCULI

The röntgenographic indications of gall-stones are direct or indirect. Direct evidence consists of a characteristic localized area of increased density, corresponding in size and shape with a calculus. Indirect evidence is afforded by the distortion of the adjacent hollow viscera by adhesions from an accompanying cholecystitis (Figs. 1 to 6). The indirect evidence will be considered first because of its greater importance. Moynihan states that it is now a matter of general acceptance that gall-stones are caused by infection.[4] If this is true, the indirect evidence is more valuable than the direct evidence, and indeed my experience has borne out this theory. In 30 per cent of the cases the lesion was detected solely by indirect evidence, and in 60 per cent of the cases the indirect evidence was a most important factor. I believe that the deformity of the cap and pars pylorica or the kinking and constriction of the hepatic flexure are stronger röntgenologic indications for surgical procedure than the presence of a calculus in the gall-bladder, without evidence of adhesions involving the hollow viscera. In such cases it is immaterial whether the infection is due to gall-stones, typhoid, or some unknown cause, or whether it has been of sufficiently long standing to allow the formation of calculi. In view of the numerous post-mortem discoveries of gall-stones, which did not cause symptoms sufficiently severe to justify surgical interference, it is obvious that the important point to determine in cases of unimpacted calculi is whether the stones are associated with cholecystitis extensive enough to cause symptoms. The accompanying adhesions manifest themselves in an

[2] Fortschr. a. d. Geb. d. Röntgenstrahlen., 1911, xvi, 335.

[3] Arch. Röntg. Ray, Lond., 1913, March, p. 374.

[4] "An address on gall-stones." Brit. M. J., 1913, No. 2714, 8.

[1] Since presenting this paper before the Atlantic County Medical Society on May 9th, Dr. Case, of Battle Creek, has published an article on gall-stones. Arch. Röntg. Ray, 1913, September.

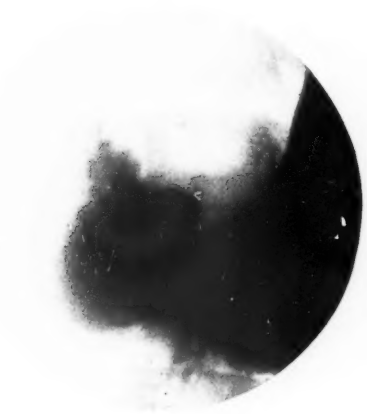


Fig. 1.

Fig. 2.

Fig. 3.

Fig. 4.

Fig. 1. Contraction of cap and pars pylorica by adhesions from cholecystitis with calculi invisible röntgenographically. Proven by surgery.

Fig. 3. Indentation in greater curvature, in which lay small, atrophic gall-bladder, containing calculus invisible röntgenographically. Proven by surgery.

Fig. 2. Absence of cap. Sphincter widened on lesser curvature. Cholecystitis with calculi invisible röntgenographically. Proven by surgery.

Fig. 4. Adhesions of cap and pars pylorica from cholecystitis. No calculus visible röntgenographically. Surgery imminent.

alteration of the lumen of the stomach, cap, or hepatic flexure of the colon.

DIRECT EVIDENCE OF BILIARY CALCULI

Regarding direct evidence of gall-stones, two things must be considered: first, the detection of the calculus, and second, its differentiation from a renal calculus or some other calcareous body in this region, especially a calcified costal cartilage. The possibility of detection depends upon the percentage of mineral salts in the stone, or surrounding a cholesterin nucleus, upon the thickness and density of the surrounding tissue, and the penetration of the X-ray tube. A soft tube, a very short exposure, and the use of an intensifying screen will give a wealth of detail impossible to obtain by ordinary methods. In-

Fig. 5.

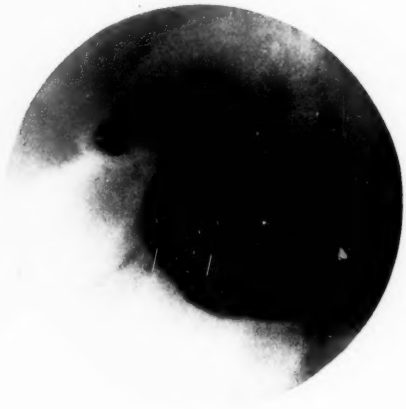


Fig. 6.

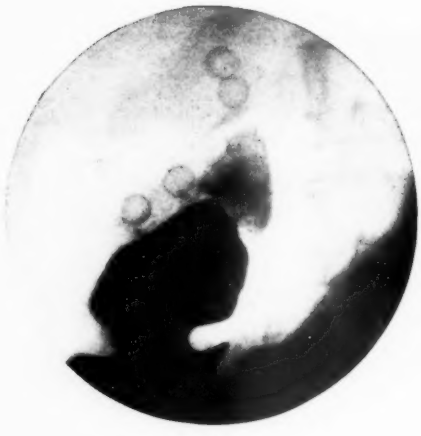


Fig. 7.

Fig. 8.

Fig. 5. Adhesions of cap, pyloric sphincter, and pars pylorica, from cholecystitis with a calculus visible röntgenographically (see Fig. 24). Proven by surgery.

Fig. 7. Pressure on terminal wave and cap by four gall-stones. Anterior view. Proven by surgery.

Fig. 6. Adhesions of cap from cholecystitis with a calculus visible röntgenographically.

Fig. 8. Same case as Fig. 7. Posterior view.

testinal content is often thus accentuated, and must not be mistaken for gall-stones. I have not found that the twisted position of the patient, claimed by Pfahler as an essential to the diagnosis of gall-stones, is of any particular advantage.

In some instances the calculus is shown so clearly that there is absolutely no doubt of its presence. In other cases it is so indistinct (Fig. 22) that it can be discovered only by making several röntgenograms, superimposing two or three of them, and examining them carefully against the sky. A stereoscopic study of two röntgenograms made with the tube in the same position or at slightly different angles sometimes aids very materially in the detection and interpretation of doubtful shadows. Occasionally an accumulation of

gas, or air artificially injected into the colon, will accentuate the stone in a remarkable manner. The organs surrounding it are relatively dense to the X-rays. There are stones which are no more opaque to the rays than the tissue which surrounds them, and there are others which are as opaque as the densest renal calculus. Between these two extremes are stones of all degrees of density. The technique above described will enable one to detect a relatively large percentage of biliary calculi.

Differentiation between biliary and renal calculi. The differential diagnosis of biliary calculi from right renal calculi is of great importance, and should be carefully made in every case presenting a stone in this region. The following points will aid in identification:

First. Biliary calculi show more distinctly and appear smaller when the plate is placed on the abdomen than when the plate is placed on the back. The opposite is true of renal calculi. When the biliary calculi is well back in the cystic duct, this variation of size is not so great as when it is in the gall-bladder. In such cases it is wise to place a small triangular marker on the abdominal wall and make two exposures, either on the same plate or on different plates, shifting the tube five inches between the exposures. Where two plates are used, match the shadows of the lead marker and measure the distance between the two shadows of the gall-stone. In order to determine with accuracy whether the shadow is nearer the anterior or posterior wall of the abdomen, repeat the above process with the plate posterior, and compare the distance between the shadows.

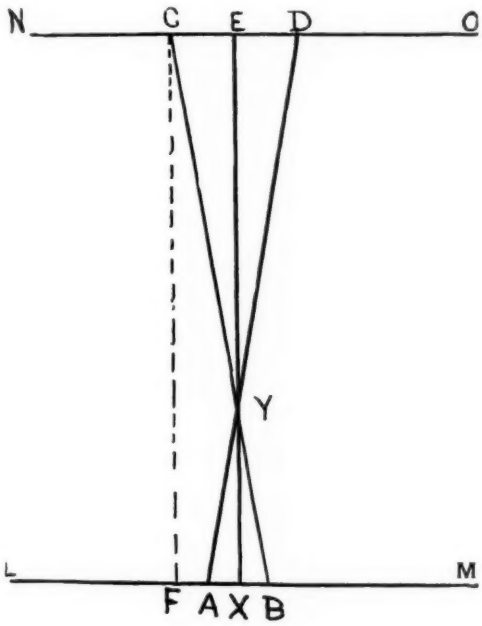
To determine distance of calculus from abdominal wall. By measuring the distances of the tube from the plate, and how far the tube is shifted between the exposures, the old principle of localizing foreign bodies described may be applied to find the distance of the calculus from the anterior abdominal wall.

Let NO = plane of the two positions of the tube.
C and D = two positions of the tube.
CD = 5″, distance between the positions of the tube.
LM = plate, parallel to plane NO.
X = shadow of marker on plate.
A = shadow of stone when tube is at D.
B = shadow of stone when tube is at C.
EX = 20″.

To find XY:—

By measurement AB is found to be 1″
$\therefore AX = \frac{1}{2}''$.
$CD = 5''$.
$\therefore DE\ 2\frac{1}{2}''$.
Lay off FX equal to EC.
△ BFC and △ BXY are right-angled △'s, and have a common acute < BCF.
The sides of right-angled triangles having a common acute angle have the same ratio.
$\therefore$ FB : FC :: XB : XY, or 3″ : 20″ :: ½″ : XY
$XY = 3\frac{1}{3}''$.

Q. E. D.



Second. The second differential point is the ring-like shadow cast by a biliary calculus when there is a calcareous coating to a cholesterin nucleus. Renal calculi seldom if ever have this appearance.

Third. When three or more biliary calculi are present they are likely to have faceted surfaces, which are readily recognized röntgenographically. If more than one renal calculus is present one is usually larger than the others, and the group frequently has the appearance of a large, branching phosphatic calculus.

Fourth. Moving the tube from side to side alters the relation of a biliary calculus to the kidney, but it does not alter the relation of a renal calculus to the kidney.

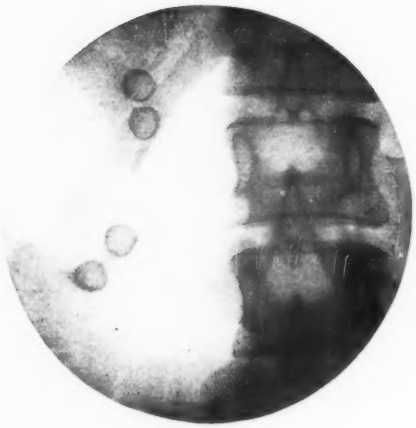


Fig. 9.

Fig. 10.

Fig. 11.

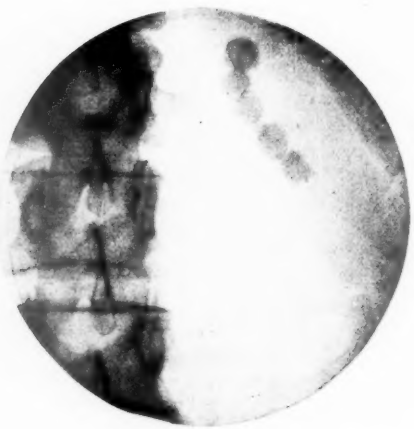


Fig. 12.

Fig. 9. Same case as Fig. 7, five days afterwards. Showing change in relative position of stones. Anterior view.
Fig. 10. Adhesions of cap and pars pylorica from cholecystitis with five calculi. Anterior view.
Fig. 11. Same case as Fig. 10. Anterior view.
Fig. 12. Same case as Fig. 10. Posterior view.

Differentiation from duodenal ulcer. Adhesions from cholecystitis so closely resemble adhesions and cicatricial contraction from prepyloric or post-pyloric (duodenal) ulcers that it is sometimes difficult to differentiate between them. But if the lesion is sufficiently extensive to indicate surgical interference, the differentiation is of more scientific interest than practical value. The points of differentiation between these conditions are as follows:

"Gall-bladder infection is usually more extensive. It involves the greater curvature and draws the stomach to the right, causing an angulation of the cap. The cap may be involved in the adhesions, but not more so than the pyloric end of the stomach. There is no evidence of a localized area of induration of the cap, and obstruction of its lumen is not as frequent as in cases of duodenal ulcer."

Fig. 13.

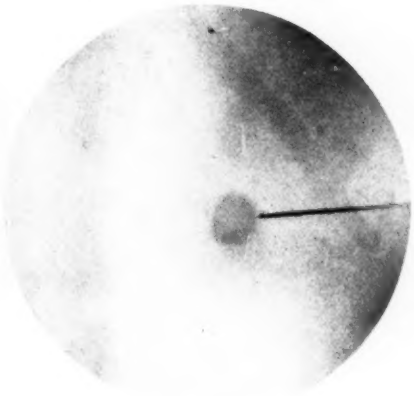


Fig. 14.

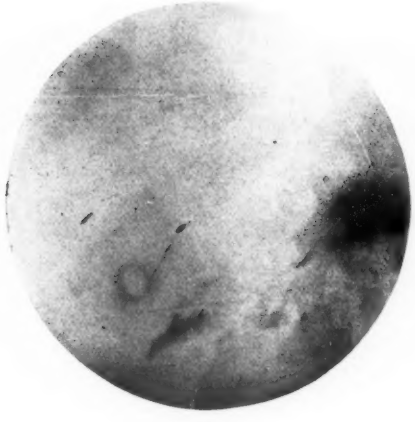


Fig. 15.

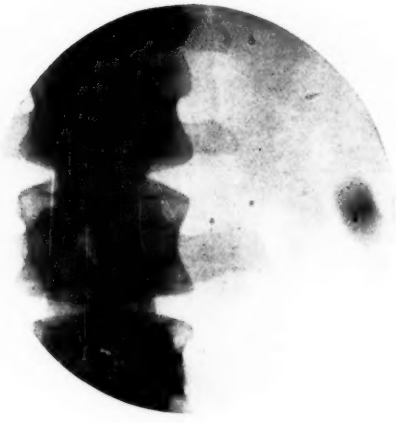


Fig. 16.

Fig. 13. Gall-stone, more dense near periphery than in center.

Fig. 15. Ring-like appearance of gall-stone, having center of cholesterin in a calcareous shell.

Fig. 14. Gall-stone, more dense near periphery than in center.

Fig. 16. Gall-stone, unusually dense to X-rays.

CASE 1. March 19, 1912. This is the most interesting of all the cases. The patient had typical symptoms of renal calculus, except blood in the urine, and was referred for an examination of the kidneys. The röntgenograms showed the region in question with sufficient distinctness for a negative diagnosis of renal, ureteral, and vesical calculus, and I advised a gastric examination. This revealed a permanently contracted cap and adhesions involving the extreme pyloric end of the stomach. I was unable to decide whether these adhesions were caused by duodenal ulcer or gall-bladder infection, but stated that surgical interference was justifiable, solely on the röntgenographic findings. Dr. Brewer, who operated, was rewarded for his confidence in the röntgenologic diagnosis by finding a cholecystitis with a number of small calculi not shown in the radiograph (Fig. 1).

CASE 2. December 21, 1910. There was a definite indirect evidence of adhesions involving the cap and pyloric end of the stomach, but there was no direct evidence of gall-stones. Some doubt existed as to whether the adhesions were caused by ulcer or gall-bladder infection, but there was no question as to

Fig. 17.

Fig. 18.

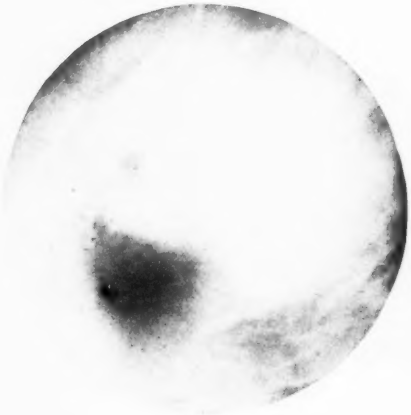


Fig. 19.

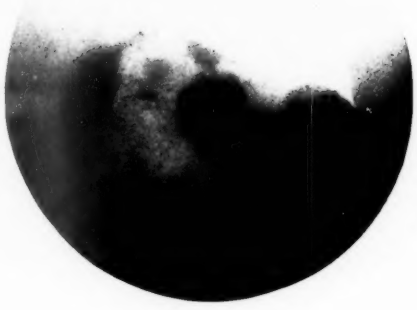


Fig. 20.

Fig. 17. Two small gall-stones, accentuated by an overlying accumulation of gas in colon.

Fig. 18. Large gall-stone, one inch in diameter.

Fig. 19. Cap so contracted by cholecystitis that only its base on left side is visible. Calculus in close proximity. Anterior view.

Fig. 20. Same case as Fig. 19. Lateral view showing how descending duodenum encircles calculus.

the advisability of surgical procedure. The lesion was found to be a cholecystitis with gall-stones, which were not shown röntgenographically (Fig. 2).

CASE 3. January 23, 1912. In this case there was no direct evidence of gall-stones, but there was a constant deep indentation on the greater curvature near the pylorus, resembling a small hour-glass stomach (Fig. 3). I thought that this lesion was caused by an old ulcer, and advised surgical procedure, which revealed a small calculus about the size of an almond in a contracted gall-bladder adherent to the gastric wall. This case has been frequently quoted as one in which I made an erroneous diagnosis. As a matter of fact, I recognized the location and extent of the lesion and stated that surgical procedure was indicated; and I maintain that recognition of the cause of the lesion is of more scientific interest than practical value.

CASE 4. January 10, 1913. The patient had had a perfectly typical attack of cholecystitis, with temperature, local tenderness, and a marked degree of jaundice. The röntgenograms showed a contracted cap and irregularities of the sphincter and lesser curvature of the pars pylorica, which constitute definite indirect evidence of adhesions involving this region (Fig. 4). The patient recovered from this

Fig. 21.

Fig. 22.

Fig. 23.

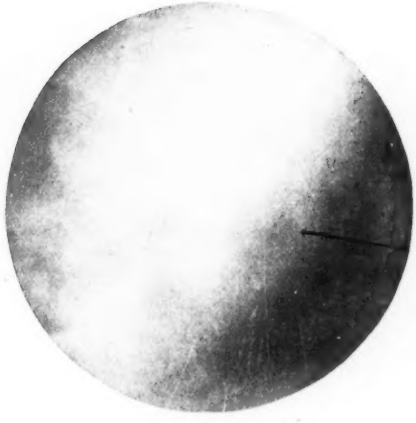


Fig. 24.

Fig. 21. Calcareous plaque in under surface of liver, diagnosed röntgenographically as a gall-stone. Proven by surgery.

Fig. 23. Gall-stone. No evidence of accompanying cholecystitis.

Fig. 22. Two indistinctly defined gall-stones. Proven by surgery.

Fig. 24. Large, faintly outlined gall-stone. Proven by surgery. For indirect evidence in this case see Fig. 5.

attack, and has not as yet consented to surgical procedure.

CASE 5. March 19, 1912. Patient shows both direct and indirect evidence of gall-stones, although the indirect is of more diagnostic importance than the direct evidence. An indistinct shadow of a large biliary calculus was present in all the radiographs (Figs. 5 and 24), but the adhesions caused by the cholecystitis were so extensive that they made me suspicious of carcinoma. The following is a quotation from the röntgenologic report.

"The absence of the duodenum, the contracted cap, the irregular worm-eaten appearance at the pylorus, and the absence of the pyloric sphincter indicate a lesion in this region which calls for surgical procedure. This lesion is probably adhesions from gall-bladder infection, probably with a calculus; but considering the irregular worm-eaten appearance of the pyloric end of the stomach, I do not feel justified in eliminating carcinomatous degeneration."

Six months later a note received from the patient's husband stated that she had not consulted a surgeon: "By following a strict diet for two months she is

greatly improved in health; while she is not considered permanently well, there is no longer any particular disturbance of the digestion or stomach."

Three months after this her physician wrote me that the patient had been seized with a sudden attack similar to the two previous attacks, which suggested gall-stone trouble very strongly. She was at once operated upon and a large gall-stone was found in the intestinal lumen, having worked through the adhesions from the gall-bladder and, at the time of this last attack, caused perforation of the bowel and acute septic peritonitis, resulting in the patient's death.

CASE 6. January 11, 1912. Both direct and indirect evidence of a biliary calculus were present, the stone resembling the one shown in Case 9: but it lies so far back in the cystic duct that there is not so much variation between its size in the anterior and posterior röntgenograms as in that case. As the characteristic ring-like appearance and the faceted surfaces were wanting, it was difficult to differentiate it from renal calculus. There were, however, indirect evidences of an accompanying cholecystitis with adhesions involving the cap and pylorus. The distended gall-bladder shows distinctly. The case was studied for a year, making two or three sets of röntgenograms, and during that time the adhesions involving the cap increased and caused a gastric retention. Surgical procedure revealed a gall-bladder corresponding in size and shape with the röntgenographic findings, the gall-stones, and the accompanying cholecystitis with adhesions (Fig. 6).

CASE 7. January 25, 1912. This case illustrates direct röntgenographic evidence of four gall-stones having the characteristic ring-like appearance. They show much clearer and appear smaller in the anterior than in the posterior röntgenogram. The clinical diagnosis prior to the röntgenographic examination was duodenal ulcer. Surgical procedure revealed a carcinoma of the appendix in addition to the gall-stones. Note the absence of adhesions, as evidenced by a normal cap and pyloric sphincter. By making a large series of röntgenograms we were able to obtain stereoscopic plates showing the manner in which the chain of calculi wrapped around the head of the pylorus (Figs. 7, 8 and 9).

CASE 8. January 13, 1911. A perfectly characteristic group of five calculi with faceted surfaces is shown in this case. The relation of these calculi to the junction of the cap and descending duodenum and their variation in size in the anterior and posterior radiographs are the pathognomonic radiographic indications of gall-stones (Figs. 10, 11 and 12). So far as I know this woman has not been operated, but there is no doubt of the diagnosis in this particular case.

CASE 9. January 11, 1910. The variation in size of a calculus in the anterior and posterior röntgenograms are shown in Figs. 13 and 14. This calculus did not have the characteristic ring-like appearance or faceted surface, but shifting of the tube between exposures altered its relation to the kidney. The patient was an unfavorable surgical risk, and no operation was performed, and no examination made for indirect evidence.

CASE 10. July 1, 1911. The characteristic ring-like appearance of a calculus, which is much more distinct in the anterior than in the posterior röntgenogram (Fig. 15), was present in this case.

CASE 11. December 11, 1905. This patient was examined for renal calculus. A distinct shadow was shown, slightly below the normal position of the kidney (Fig. 16). Operation failed to reveal a renal calculus, but a biliary calculus corresponding in size and shape with the röntgenographic finding was found.

CASE 12. May 13, 1913. The clinical history was one of acute attacks of pain, which had been diagnosed both as gall-stone and ptomaine poisoning. The indirect evidence showed a persistent contraction of the cap and an irregularity of the pyloric sphincter, believed to be either a spasm or adhesions. Subsequent plates, made for direct evidence, showed two clear-cut, well-defined shadows having the appearance of diplococci. They were visible in several plates, but happened to be very much accentuated in one of the plates presenting an accumulation of gas in the overlying gut (Fig. 17).

CASE 13. May 22, 1913. For two years the patient had had occasional attacks of severe pain and upward pressure in epigastrium, lasting for six and seven hours and leaving him sore and weak. He had lost thirty pounds in about a year. The röntgenograms showed direct evidence of a large calcified body anterior to the kidney and directly in the position of the gall-bladder (Fig. 18). The indirect evidence was pylorospasm and contraction of the cap, with a possibility of some adhesions. This is a recent case, and the patient is waiting until the summer is over before submitting himself to surgery.

CASE 14. May 15, 1913. The röntgenograms (Figs. 19 and 20) show both direct and indirect evidence. The cap is contracted and the pyloric sphincter irregular. Röntgenograms made in the lateral direction show that the descending duodenum encircles a gall-stone.

CASE 15. October 4, 1912. The direct and indirect evidence were at variance in this case (Fig. 21). The direct evidence showed a calculus, which I interpreted as a gall-stone after a most careful examination, including an application of all of the methods previously described. The indirect evidence showed no adhesions, as there was a perfectly normal stomach, pylorus, and cap, freely movable. Surgical procedure failed to reveal a biliary calculus, but there was a calculus in the under surface of the liver in close proximity to the gall-bladder, which was involved in slight adhesions, not, however, extending to the capsule in the liver. A thick band of the omentum was attached to the stump of an appendix previously removed.

CASE 16. February 26, 1913. The plates showed such slight ring-like shadows that they could be perceived only by viewing the plate against

the sky, and it was difficult to differentiate them from calcified costal cartilages (Fig. 22). We traced the outlines of two complete and several incomplete rings, and assumed that there were three or four calculi. Surgical procedure revealed only two calculi and a mass of thick tarry bile.

CASE 17. February 15, 1913, shows a calculus in exactly the proper relation to the duodenum and hepatic flexure for a gall-stone; but the patient was not a good surgical risk, and the symptoms were not sufficiently severe to indicate surgical procedure (Fig. 23).

CASE 18. March 24, 1911. A calcareous body was present in a series of röntgenograms. The clinical symptoms were those of gall-stones. Surgical procedure revealed a gall-stone; but there was also a calcified gland palpable in the field of operation, so it is impossible to state whether or not this really was a gall-stone.

CASE 19. February 26, 1912. The findings in this instance were almost identical with those of Case 18. Patient has not yet been operated on.

CASE 20. October 6, 1908. The plate illustrating this case was loaned to the surgeon and lost track of. The diagnosis was that of a gall-stone a little larger than a pea, one-half inch to the outer side of the median line, between the eleventh and twelfth ribs, behind and opposite the end of the sixth rib in front. The surgical findings confirmed the diagnosis.

Indirect evidence of gall-stones, or rather of an accompanying cholecystitis, as presented by adhesions involving the stomach, cap, duodenum, or hepatic flexure, is of more clinical value than the detection of the gall-stones themselves, because the adhesions represent an accompanying infection, requiring surgical intervention, while a gall-stone without infection may remain in the gall-bladder indefinitely without causing symptoms.

In conclusion, I would state that gall-stones may be detected sufficiently often to justify a röntgenographic search for them, but the absence of any direct evidence does not justify one in making a negative diagnosis, and should not prevent surgical intervention provided it is clearly indicated by the history.

Above all, I plea for a differential diagnosis between right renal calculi and gall-stones.

A FURTHER STUDY OF PERICOLONIC MEMBRANES

By JOSEPH RILUS EASTMAN, M. D., Indianapolis

PERICOLONIC membranes, or pseudoperitoneums, present certain rather definite variations in structure, form, position, and relations. However, a correct classification of these formations can hardly be made with our present insufficient and disconnected data relating to their nature and origin. For the convenience of study these anomalous membranes may be divided as follows:

Group 1. Those peritoneal sheets or ribbons which, because of their position and attachments, seem to represent exaggerations of normal anatomic fusions, folds, or ligaments; for example:

(*a*) Those representing in an exaggerated or modified form.—The phrenocolic ligament of Toldt, the costocolic ligament, the hepatocolic ligament, etc.

(*b*) Those representing in an enlarged or modified form.—The bloodless fold of Treves.

(This is not always bloodless. It may present parallel or irregularly disposed blood-vessels).

(*c*) Those representing in amplified form.—The fold of Reid (genitomesenteric fold, or Lane's band).

(*d*) Those representing in amplified form.—The crescentric upfoldings of serosa, which divide the space lateral to the ascending and descending colon into peritoneal fossæ.

(*e*) Those representing in amplified form.—The attachments of the great omentum to the colon, occasionally resulting in fusion of the omental serosa to the peritoneum of the colon throughout its length.

Group 2. Pericolic membranes which correspond in form and position to anomalous peritoneal fusions or folds which appear frequently in the fœtus, but which are not constant and therefore not anatomic, as:

(*a*) The parietocolic fold of Jonnesco. This is believed by Eisendrath, Greensfelder, and Fallon to be a normal anatomic fold.

(*b*) The several so-called root folds.

(*c*) Nondescript peritoneal folds about the appendix and caput coli.

(*d*) Adhesions of the peritoneum of the colon to the upfolding peritoneum of its own mesentery (Robinson and Reid).

(*e*) A blanketing of the colon by the fused anterior and posterior peritoneal laminæ of the great omentum. The fat of the omentum is absent. It may be replaced by an abundance of parallel blood-vessels disposed, as a rule, transversely to the colon.

Group 3. Pericolic membranes occurring anywhere upon the large intestine, frequently over the splenic angle, which represent reaction to irritation of inflammatory or other character occurring in post-natal life.

(*a*) Membraniform adhesions occurring about the colon, the result of mild recurring peritonitis, often secondary to chronic colitis (Virchow and Gerster). These are similar to the pseudoperitoneums seen occasionally upon the broad ligament, the uterine fundus, and the uterine adnexæ. I have produced such formations in rabbits by prolonged administration of morphine and by anal ligation inducing coprostasis.

(*b*) Changes in the peritoneum of the colon due to pathology of the circulation, as hyperæmia or varicosities (Hertzler and Cuneo).

All of the varieties of pericolic membranes enumerated above have been observed in the fœtus, and whatever the fundamental processes of pathology which cause these formations may be, it seems reasonably certain that those processes are often active in fœtal life and that all forms of pericolic membranes, with the possible exception of those embraced in Group 3, can be traced to fœtal fusion.

The failure to observe membranous pericolitis in young children has been interpreted with some reason as evidence showing that such membranes cannot be associated in any way with fœtal peritoneal anomalies, but on the other hand must, in practically all cases,

be consequent upon certain agencies which do not become operative before maturity.

It is true that membranous pericolitis has been observed much less frequently in children than in adults, but there are several quite obvious reasons for this. Abdominal operations for all causes are much less frequent in children than in adults. The simple controlled diet of children, their free indulgence in exercise, and other healthful habits of living protect them in some measure from the clinical process which pericolic membranes induce. If the clinical process be established in a child, neurasthenia, one of the conspicuous symptoms of pericolitis, is not always a part of it, therefore the condition may go undiagnosticated and unoperated. It is a mistake, however, to assume that membranous pericolitis does not occur in young children.

With a view to considering the collective experience of men who have large opportunities for observation in this field, and thus perhaps to shed some light upon the question of the incidence of pseudoperitoneums in children, as well as upon the subject of their nature and origin, communications of inquiry were addressed to representative teachers of surgery in this and other countries. Upon examination of the replies it appears that pericolic membranes have been observed during abdominal operations upon children by nearly one-half of the surgeons addressed. By some of those who replied in the affirmative, the pericolic membranes were assumed to represent congenital sheets or bands as described by C. H. Mayo and Flint. By others the membrane-forming pericolitis was looked upon as a condition secondary to colitis with subsequent mild repeated peritonitis, in the sense of Gerster and Pilcher. Others believe the membrane to be due to a combination of the causative factors mentioned above. However, all of the following have found anomalous pericolic membranes in children of twelve years of age, or younger: Roux, Stiles, Czerny, Albrecht, Lane, Willy Meyer, C. H. Mayo, M. L. Harris, Flint, Binnie, McArthur, Gerster, Greensfelder, Hill, Summers, Knott, Coffey, Connell, Porter, Eisendrath, Hertzler, Finney, Sherman, Seelig, Collins, Cheever, Freeman, and Haines.

Often in young persons Professor Roux has observed a veil of new and almost free peritoneum containing young blood-vessels disposed transversely. The symptoms in these children were like those of chronic appendicitis. Roux observed these membranes at various levels upon the ascending colon. Stiles of Edinburgh has observed pericolic membranes in children from a few years of age upwards. Czerny has frequently seen anomalous peritoneal membranes upon the colon in children. M. L. Harris observed a typical Jackson's parietocolic membrane in a boy of twelve, and Willy Meyer, C. H. Mayo, D. N. Eisendrath and L. L. McArthur have frequently observed Jackson's membrane in young children. Flint observed the membrane in children eight, ten, twelve and sixteen years of age and believes that they occur in the same incidence in children as in adults, the reason that they are not more frequently seen being due to the fact that we operate upon adults ten times more frequently than upon children.

Notwithstanding the reports of the surgeons mentioned above, which give instructive evidence as to the not infrequent occurrence of membranous pericolitis in children, it may be said that, before conclusions are drawn as to the incidence of Jackson's membrane in children, such clinical evidence should be supported by autopsy reports covering many examinations of the peritoneum in children. Flint supplies such supporting testimony received by him from Dr. Samuel Harvey, instructor of pathology in Columbia University. Harvey did 108 autopsies on infants from one to two years of age. In 16.6 per cent of the cases he found membranes of the parietocolic type. In 13.9 per cent of cases he found a membrane covering the caput coli. Lane's band, or Reid's genitomesenteric fold was found in 9.3 per cent of cases. Flint says that the work of Harvey supplies the missing link between the fœtal structures and the adult veils, and confirms absolutely their congenital origin, and adds that he has no reason to change his views as expressed in his paper published in the *Johns Hopkins Bulletin* for October, 1912, in which he stated his conclusion that pericolic membranes of adults are

Fig. 1. In center of picture a pericolonic membraniform adhesion. Induced experimentally in a rabbit by anal ligation.

instances where the secondary fusions of the peritoneum in embryonic life are more extensive than we are usually accustomed to consider the norm.

In 500 autopsies on children under six years of age Albrecht of Vienna found pericæcal membranes present in about 15 per cent of the cases (Fallon[1]).

Van Buren Knott reports the cases of two infants, one year old, operated for intussusception, who had well-developed Jackson's membranes.

Eisendrath has found pericolic membranes in children of all ages from four to sixteen, and believes them to be of frequent occurrence in children. He believes, moreover, that such pseudoperitoneums are fœtal peritoneal folds which under some circumstances, like the normal pleura or any other serous membrane, become infected and thickened, and then cause kinking and other forms of chronic obstruction.

C. H. Mayo, Harold Stiles, Sir William Bennett, and others express the belief that membranous pericolitis must be of rather infrequent occurrence in young children, and this view is borne out by the experience of many surgeons who, despite very great opportunities for observation, have not seen a case of Jackson's membrane in a child. Some of them (Tinker, Schwyzer, and others) look upon uncomplicated pericolic membranes as quite harmless deviations from the normal and, having no special interest in them, do not seek them as a routine practice. Nevertheless, the negative reports as to the incidence of membranous pericolitis in children are not without meaning in view of the quality and volume of experience represented by the following, who have not observed pericolic membranes in young children: von Eiselsberg, Maylard, Handley, D'Arcy Power, Pierce Gould, Kellock, Wilms, Verhoogen, Pilcher, Crile, Walker, Bovée, Peterson, Montgomery, Morris, Murphy, Jackson, Ries, Deaver, Rodman, McMurtry, Kanavel, F. H. Martin, Bartlett, J. S. Horsley, Littig, A. T. Mann, A. A. Law, Grant, Moore.

It should be remembered, however, that these men are surgeons and, therefore, would as a rule observe only such pericolonic membranes as are associated with clinical manifestations.

Several others who replied in the negative are gynecologists of large experience, but who are called upon very rarely to make abdominal operations in young children. Verhoogen, of Brussels, speaks of the possibility of his having overlooked pericolic membranes in childhood for the reason that his attention has never been directed to them. Among those who had not encountered anomalous pericolic membranes in children, three remarked that they were doubtless present in childhood, but since it is the infective accidents of later life which bring them into the clinical field, they escape observation in children.

A few of those who do not approve the hypothesis of embryonal origin of any variety of pericolic membrane report the frequent

[1] Boston M. & S. J., 1913, Oct. 23.

finding of Jackson's anomalous membrane in association with cæcum mobile. However, this combination in itself furnishes evidence suggesting a fœtal origin, for in the mobile cæcum swinging as it does through an abnormally large arc, we have an instance in which a secondary fusion between the cæcum and the mural peritoneum may be drawn out readily in a degree and form quite beyond the norm.

Although it necessitates a digression from the subject-matter of this paper, it may not be amiss to call attention to another phenomenon which suggests a fœtal origin of pericolic membranes about the caput coli. It is the almost constant observation that when during operation for removal of the appendix vermiformis the uninflamed appendix is found buried under a pericolic membrane of the exaggerated Treves' bloodless fold variety, such a veiled and pocketed appendix is almost always of fœtal type — that is, long and very slender. Such membrane-covered appendices are often not more than two millimeters in diameter and are rarely of a diameter exceeding three millimeters and show many evidences of interrupted development.

Almost a score of surgeons, including Stiles, Mayo, Verhoogen, Vander Veer, Connell, Eisendrath, Summers, and McArthur, answered the inquiry as to the origin of the membranes by stating their belief that they are developed in the fœtal period.

A somewhat smaller number, including Handley, Czerny, Crile, and Gerster, incline to the belief that membraniform pericolitis is the consequence of a colitis spreading through to the peritoneal surface.

A third group, including Pilcher, Morris, Finney, Jackson, Hill, Kanavel, Ries, and Horsley, believe pericolic membranes, as seen in the adult, to be in part congenital and in part acquired as a result of extension of inflammatory processes within the lumen.

Gerster, recognizing the part which persisting fœtal folds may have in adult intestinal pathology, makes a sharp distinction between membraniform anomalies representing developmental accidents and those essentially different membranes which are consequent upon colitis. He has frequently found the two varieties in association, and believes that

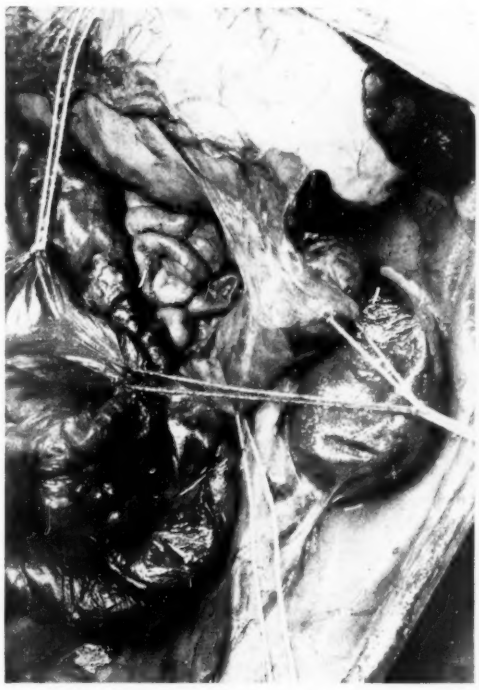


Fig. 2. Fusion between colon at splenic angle and omental serosa producing a pericolonic membrane. Result of anal ligation in a rabbit.

the congenital bands frequently represent the starting point of similar pathologic formations of adult life.

Several surgeons in their answers volunteer the suggestion that pericolic membranes about the caput coli are more often the cause of appendicitis than the effect, torsion, angulation, or compression of the appendix by the membrane so interfering with drainage as to lead to inflammation.

There would have been, without much doubt, less divergence of opinion as to the absolute and relative frequency of pericolic membranes in children if the questions and answers had been formulated with a definite understanding as to what really constitutes a Jackson's veil as distinguished from a membranous adhesion.

Anomalous peritoneal folds, wherever and whenever found, notwithstanding their variations in macroscopic and microscopic appear-

ance, are with respect to their nature and origin probably quite closely related. The frequency of anomalous pericolic folds in the fœtus and the fact of their very common presence in young children indicate that, whatever the fundamental pathologic process which causes them may be, this process frequently becomes operative in embryonal life.

Concerning pericolic membranes which arise during adult life it seems fair to suspect, in view of experimental evidence, that they result, for the most part, from the adhesions of a plastic peritonitis, usually of mild chronic character. Membranes thus formed are usually thinner and less vascular than those which date from the fœtal period, and, as remarked by Lardenois,[1] do not resemble embryonal peritoneum.

The writer has experimentally produced such pericolic membranes in rabbits by inducing a low grade plastic peritonitis. The mild adhesive peritonitis occurred as the result of the plugging of the large intestine with its own contents. Extreme coprostasis was induced by intermittent ligation of the anus and administration of morphine over a period varying from two weeks to three months. Care was taken not to confuse the normal long-flowing mesenteries of the rabbits with membraniform adhesions. As in other quadrupeds the mesenteries of the rabbit are luxuriantly developed, a single mesentery in some cases enveloping and supporting several intestines. The membranous adhesions which were attributed to the artificially induced plastic peritonitis occurred in cases in which the intestines were held together over extensive areas by plastic lymph. The membranes were brought to view by separating the intestines at the site of imperfectly organized adhesions.

Such pericolic membranes are not different in histology and origin from similar membranes about the uterus and uterine adnexæ, all representing reactions of the peritoneum to irritation.

Of the types of pericolic membranes produced experimentally in rabbits as mentioned above, one of the most common was that represented by a blanketing of the splenic angle with the fused anterior and posterior laminæ of the great omentum, suggesting an exaggeration of the phrenocolic ligament of Toldt.

Hertzler and Cuneo attribute those vascular pericolic membranes, with vessels coursing horizontal to each other in the line of the long axis of the ascending colon or transversely to it, to pathology of the circulation. Cuneo believes that the blood-vessels produce the membrane. It is, however, no contradiction of the position of these writers to assume that the hyperæmia or varicosity spoken of by them, as well as the insufficiency of the muscular coat of the intestine mentioned by Tuffier, suggest reactions of the peritoneum to irritation. Lenormant and Oberlin[1] emphasize the importance of the relation of adhesion in plastic peritonitis to pericolic membranes. They make a distinction, however, between plastic membranous adhesions of adult life and those of embryonal origin.

Adhesions in fœtal life probably bear the same relation to pericolic membranes as do adhesions of the adult. It is probable that the true Jackson's membrane appearing upon the right half of the colon is usually an affair of fœtal development, the result of accidents of cæcal descent and torsion. On the other hand, membraniform adhesions of post-natal life, in the sense of Virchow, Gerster, and Pilcher, may appear anywhere upon the large intestine, the most frequent site probably being the splenic angle.

In conclusion it may be said that it seems probable that several factors enter into the causation of pericolic membranes; that the several factors are closely related; that fusion or adhesion as a result of reaction to irritation, hyperæmia, or developmental accidents in the prenatal or post-natal period may lead to anomalous peritoneal formation; that in the fœtal period of development abnormal fusions occur with notable frequency and with interesting constancy as to location and form; that pericolic membranes unassociated with chronic or acute colitis or peritonitis are of fœtal origin; and further that in a great measure the same pathologic processes underlie pericolic membrane formation at all ages.

[1] J. de Chir., 1913.

[1] Rev. de gynéc. et de chir. abdom. Par., 1913, Sept.

DEPARTMENT OF TECHNIQUE

FIXATION OF FRACTURES BY MEANS OF AUTOGENOUS INTRAMEDULLARY BONE SPLINTS

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AT the present time there is probably no subject that has undergone the changes or made the advancement that we find taking place in the domain of bone surgery.

We have seen the gradual evolution of the open treatment of fractures from its inception where the splendid results of a few pioneers encouraged others to emulate their success. Possibly through inferior technique or judgment in handling their cases the results were unsatisfactory. This led to open objection by some, disagreement and doubt by others, as to the advisability of the open treatment of fractures. Such remained the status of the open treatment of fractures until the results of the successful were multiplied and the mistakes of the unsuccessful were pointed out.

We are indebted principally to Mr. Lane for a technique which, if followed, seemed to insure success. Since the advent of his methods of operation we have seen the operative treatment of fractures increase enormously and it is still increasing.

But while the use of the Lane plate is becoming more frequent, I believe the time is soon coming when we shall lay aside the Lane plate, the ivory pegs, and various other articles made use of by surgeons for the fixation of fractures and shall fix them without the use of foreign substances, i.e., by means of autogenous bone splints or transplants.

I shall consider only the use of the autogenous bone splints in these remarks because I am of the opinion the use of homogenous bone splints—those taken from another human body — kept in cold storage or otherwise is attended with so many difficulties of possible blood reactions, diseased conditions, or infections as to render its use undesirable at least for some time to come. In using a foreign bone it seems we are exposing the patient to a lot of unnecessary dangers to procure an inferior article. I believe at the present state of our knowledge all will agree that the autogenous bone graft is far superior to that taken from another individual.

Any one observing the effects of burying nonabsorbable substances in the human body has probably been struck first by the tolerance of the tissues to the foreign substance and secondly by the disturbance or untoward remote effects arising from a foreign substance which has remained quiescent in the tissues for a long time. A foreign body, Lane plate, silver wire, bone-peg, or other favored substance is used in the fixation of a fracture. The wound heals per primum. The patient is discharged well. We note a perfect result and are satisfied.

Perhaps a year elapses, perhaps more, when that patient comes back, or goes to some one else, complaining of pain at the site of the old break. A careful skiagraph shows a chronic osteitis which may subside with rest and immobilization to reappear later, or it may persist until the foreign substance has been removed, and the process may go on after the removal of the foreign body.

The fixation apparatus may be tolerated without reaction by the tissues for many years when suddenly a slightly lowered resistance in the general condition of the patient furnishes the opportunity for an endogenous infection. Certain areas of the human body are by structure and vascular supply more susceptible to infection than other portions and such a condition we create in the contacting tissues by the insertion of a nonabsorbable substance. In this position, as in the natural vulnerable areas of the human organism, infection does not necessarily follow in a certain length of time or ever for that matter, but as long as the foreign substance is embedded, there is just as surely a point of lowered resistance and potential trouble and may become a nidus for infection at any future time. For this reason the results of Mr. Lane, with his incomparable technique, have been so excellent. He recognizes the susceptibility of the traumatized tissues to any infection and averts trouble by a perfect aseptic technique.

The difference between the old operation and

Fig. 1. 1. Ratchet brace. 2. Vise. 3. Hollow auger. 4. Twist drill bits. 5. Board on which vise is fastened.

the new does not lie in the primary operation. The damage to the tissues has been done when the fracture occurred. The operation is the same in all cases and presupposes a perfect aseptic technique. Primary liability to infection is the same in both cases, but the difference does lie in the late manifestations of trouble arising from a buried foreign substance on one hand and the autogenous bone splint on the other hand soon becoming an active, living, integral part of the bone in which it is inserted. It requires no removal; subjects the patient to but one operation; and does not exist as a menace in subsequent years.

There are several reasons why autogenous bone splints have not been more widely used in the routine fixation of fractures. Probably first and foremost among these has been the paucity of suitable instruments. Without suitable instruments the operation becomes impossible.

The operation of bone-plating achieved no popularity with the profession until suitable instruments were devised, thereby rendering the operation comparatively easy and a perfect asepsis possible. When the technique of fixation of fractures by autogenous intramedullary bone splints becomes perfected to such a state it can be performed with almost as little trouble as plating, and with as rigid an aseptic technique, I am firmly of the opinion the metallic plates, etc., will deservedly give way as the method of choice in the open fixation of fractures. Suitable instruments in the ordinary surgeon's armamentarium for this class of work are exceedingly rare. Outside of Dr. Murphy's most excellent medullary reamers and retractor-guide, the special instruments devised for this work are very few and, as far as I can discern, very unsatisfactory.

With an idea of what these instruments should be I looked about and found a very satisfactory embodiment of my ideas in the tools of the carpenter and carriage maker. I believe that any one with a fair amount of mechanical ingenuity and with sufficient practice on the cadaver, or on other bone, to familiarize themselves thoroughly with adjustment and use these instruments, will find them very satisfactory. They consist of:

(*a*) Ratchet brace, similar to those used by carpenters or wood workers;

(*b*) Twist drill-bits, about eight or ten in number in graded sizes from about one-fourth inch in diameter to five-eighths inch in diameter;

(*c*) One small vise with clamp to fasten to board — the vise is a small affair weighing about one and one-half to two pounds;

(*d*) One adjustable hollow auger, such as is used by carriage makers for fashioning the ends of spokes that go into the felloe. It can be adjusted to cut tenons any size from one-fourth inch to one inch in diameter and upward.

(*e*) A piece of ordinary board of suitable length to be placed in sterilizer and of proper thickness for the attachment of the clamp of the vise.

The technique is very simple. For instance, if it is desired to insert a transplant in a simple fracture of the tibia: A linear incision is made over the tibia, the ends elevated above the skin one at a time, a drill bit selected which will just clean the medulla out to solid bone. This drill bit is put in the brace and the ratchet on the brace turned so the drill will turn in the right direction. The handle of the brace cannot be turned entirely around on account of the proximity of the leg, hence the necessity of the ratchet brace. The medulla is reamed for such distance of one or two inches as is necessary to secure a firm socket for the transplant. The end is then dropped and the other end treated in the same way. The size of the bit is then noted and a transplant cut from the crest of the opposite

tibia by means of a drill and chisel, cutting a little larger than the transplant required. The vise is fixed to the board and this held firmly on the instrument table by an assistant. The transplant is picked up by means of forceps and one end of it is grasped by the jaws of the vise. The hollow auger is then put in the brace and by means of the screw is adjusted to cut a tenon the exact size of the drill bit used in excavating the medulla. The size of the tenon is shown by the gauge on the face of the hollow auger. The length of the tenon should equal the combined depth of both of the holes reamed. With the bone held in the vise, in a few moments the transplant can be converted into a perfectly smooth, true, living bone tenon of exactly the right size to fit the hole in the medulla. In the ordinary fixation of a simple fracture a long transplant is not necessary nor desirable. A transplant of one and one-half or two inches, provided it fits accurately, is sufficiently long to hold in perfect position a fracture of femur, humerus, etc.

If a long transplant is required— more than four inches— it will be necessary to true up one end of the transplant as far as the hollow auger will cut (about four inches), then reverse the bone in the vise and true up the other end, taking care that the boring from one end meets accurately the boring from the other end. The bone is then taken by means of forceps and transferred to the place prepared for it, where it will fit perfectly if the instruments have been properly adjusted.

The Lane technique may be carried out in using these instruments with the greatest ease.

If it is desired to insert a transplant between the ends of a bone, a portion having been destroyed by accident or disease: For illustration, say there is a six-inch piece lacking in a tibia. To repair the defect a half-inch hole is reamed in the medullary canal of the upper fragment to a depth of, say, two inches. Another hole of the same diameter and three-fourths inch in depth is made in the lower fragment. A transplant is cut from the opposite tibia eight and three-fourths inches in length by about five-eighths in diameter. Placing the transplant in the vise and with the hollow auger fastened in the brace and adjusted to cut a tenon one-half inch in diameter and with the sliding gauge on side set for two inches in length, the upper end is completed. Reversing the bone in the vise and adjusting the gauge of length to three-fourths of an inch, the opposite end is completed.

Fig. 2. Hollow auger showing face with scale of diameters and screws to adjust diameter and length of tenon cut.

This leaves the large part of the transplant six inches in length — just sufficient to fill in the gap. The upper or long tenon is inserted into the hole prepared for it. It has no lateral motion, neither can it push up farther into the medulla than the two inches cut by the hollow auger. Then by making a moderate amount of traction the lower end can be pushed into the hole prepared for it. The lower tenon has purposely been made shorter to facilitate insertion.

When in place the leg will be found to have the exact length required and will be rigid.

Regeneration of the transplant is undoubtedly greatly facilitated by extensive accurate apposition to living bone cells and by the immovable fixation in such a position.

The time required to perform the operation of fixation of simple fractures by means of the autogenous graft nearly approaches that required for Lane plating. With the old method of preparing a transplant, when to secure a fit was more or less accident, coupled with the design, there is no comparison.

These instruments can all be boiled to insure absolute sterility; can be procured from any dealer in carpenter tools, and at a trifling cost.

In conclusion I desire to thank Lieutenant Col J. L. Phillips, Assistant Chief Sanitary Officer, for permission to publish this paper.

INDIRECT TRANSFUSION OF BLOOD

By ANDRE CROTTI, M. D., Columbus, Ohio
Surgeon to Grant Hospital and Children's Hospital

ALTHOUGH thoroughly familiar with the technique of vascular surgery, I have always felt that the direct transfusion of blood by arteriovenous anastomosis was a very delicate operation, and very often unsuccessful. Everyone who is familiar with that kind of work knows how difficult it is to anastomose properly, for instance, the small vein of a child with the large radial artery of a man, and even when the anastomosis has been successfully performed, there is no way of telling if the arterial blood is running into the vein, and if it does, how much has been transfused. The operation is a long and difficult one. The same objections are true for the other methods of transfusion, as for instance, when performed with small glass tubes prepared with alboline or paraffin. Therefore, I sought to find a simpler method which might be just as efficient, if not more, and become more popular on account of its simplicity. It is called indirect transfusion of blood.

With Dr. Shilling, pathologist at Grant Hospital, I made a series of experiments on dogs in order to determine the coagulation time of the blood, when withdrawn, first, in a sterile, dry glass; second, in a glass boiled in normal salt solution; third, in a glass boiled in alboline. The average coagulation time was from five to eight minutes for blood withdrawn in a dry, sterile glass, and from seven to twelve minutes for blood withdrawn in a glass boiled in salt solution or in alboline. Therefore, I thought there would be plenty of time to withdraw blood with a syringe from a vein and reinject it into another one without running any danger of coagulation. This was indeed successfully demonstrated in each of our numerous experiments. We were able, provided that certain rules were followed, to withdraw several hundreds of cubic centimeters of blood from a vein of a dog and to reinject it into the vein of another without a particle of trouble, and I was able to demonstrate the efficacy of the method in two cases which I herewith report.

Case 1. The patient, Mr. G. H., 16 years old, was referred to me by Dr. McKenzie, of Lancaster, Ohio, on April 2, 1913. In October, 1912, patient noticed a lump in his right cheek causing shooting pain in the right temporal fossa. No trouble in chewing or swallowing. No soreness. An unsuccessful attempt to remove this tumor was made by another surgeon in January, 1913.

When I saw the patient in April, I found in the right cheek below the zygomatic bone, a tumor located outside of the buccinator muscle. It was about the size of a walnut, had sharp limits, was lobulated, firm in consistency, painless to pressure and not adherent to the skin. A bidigital examination, one finger in the mouth and the other outside, showed that the tumor had an upward prolongation which disappeared under the zygomatic bone. The latter one on the right side was more prominent than on the left side. There seemed to be a slight paretic condition in the region of the facial nerve, although there was no paralysis. Tonsils and adenoids were very much enlarged. The right posterior choana was filled with a mass, red-bluish in color and bleeding extremely easily when touched with an instrument. I made the diagnosis of myxofibromatous polypus and concluded that this tumor in the cheek was only an extension of this same tumor passing through the pterygomaxillary opening. I referred this case to Dr. John Edwin Brown, eye and nose specialist, who very successfully and skillfully removed the polypus by the canine fossa route. The operation was extremely bloody, difficult, and lasted an hour. At the end of the operation the patient was more or less exsanguinated. Pupils were dilated; pulse could not be felt. Patient seemed to be in imminent danger of death. Very quickly I made an indirect transfusion to the patient of one and one-half ounces of venous blood taken from the father and injected a quart of salt solution intravenously. The blood transfusion did not last more than five minutes. The boy recuperated gradually, his pulse, although extremely rapid, became palpable; three hours after, his hæmoglobin was 55 per cent. In the hurry, unfortunately, hæmoglobin had not been taken before transfusion. Gradually the boy's strength came back and he made an uneventful recovery. The microscopical examination confirmed the diagnosis of myxofibroma.

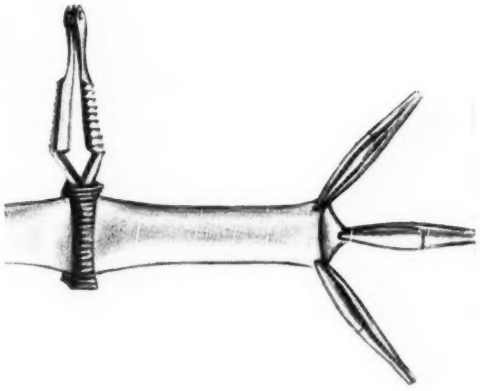
Case 2. Mrs. M. P., 30 years of age, was referred to me by Dr. H. M. Beebe, Sidney, Ohio. The patient had a goiter, examination of which showed it to involve both lobes and the isthmus of the thyroid, the lower pole of the right lobe dipping behind the clavicle and the sternum. In addition to this intrathoracic character of the goiter, the patient showed some symptoms of thyroid intoxication, so she entered the hospital, was put to bed and a preparatory treatment of several days carried out.

She came to operation in apparently good condition, although the pulse was still rapid. Morphine-scopolamine, ether anæsthetic. The right upper pole was ligated and the right lobe and the isthmus resected. The amount of hæmorrhage was only moderate but the operation offered some difficulty on account of the intrathoracic portion of the goiter. The patient woke up on the operating table, her pulse at this time being rapid and weak, but her color was good and no signs of collapse apparent. Five or six hours after, her heart suddenly collapsed and despite all treatment, including an indirect transfusion of 75 ccm. of blood from the husband, the patient died.

Respiration continued for some time after the heart had ceased to beat. This case was a typical case of heart failure.

The technique of the indirect transfusion in this case was identical to that used in Case 1, but

Figs. 1 and 2.



Mosquito forceps and artery clamp applied to vein.

the indications for the procedure in Case 1 became apparent in the operating room where it could be carried out without any appreciable loss of time; while in Case 2 the collapse occurred several hours after the operation, in the patient's room, and some considerable time was necessary to prepare, sterilize, etc., before proceeding. The patient in the second case was beyond aid, although the actual elapsed time for the indirect transfusion itself was slightly more than five minutes.

Technique. Iodine preparation of the skin of the donor. Local anæsthesia with novacaine, one per cent. Incision of the skin three or four ccm. long in the angle of the elbow. The cephalic vein is dissected out and cut at the upper end

of the incision. The proximal end is ligated with catgut. To the distal end three small mosquito forceps are applied at equal distance one from the other in order to maintain the lumen of the vein open. (See illustration.) A small artery clamp applied a few centimeters below prevents the blood from leaking.

The same operation is performed on the recipient with the difference that the vein is ligated at the lower end of the incision.

A blunt needle which has been adapted to a syringe is introduced into the vein of the donor, in the opposite direction of the blood current; blood is aspirated into the syringe, and reinjected into the vein of the recipient, in the same direction as the blood current.

The transfusion can be repeated as often as is deemed necessary without coagulation, provided needle and syringe are freshly washed each time in a warm normal salt solution. The best plan is to have two needles and two syringes and to have one set washed by an assistant while the other is in use. When transfusion is terminated, the veins are ligated and the skin incision closed. By this method any amount of blood can be transfused safely from one patient to another and the exact amount of blood transfused is known. The fact that the blood is venous seems to be without importance. My first case would seem to prove that it is not necessary to transfuse large quantities of blood to save a patient. However, it can be done if deemed necessary. The technique is simplicity itself, and can be used by anyone. A few bubbles of air are of no importance.

THE MATHEMATICAL CALCULATION OF PROGNOSIS IN FRACTURES AT THE ANKLE AND WRIST

By E. H. SKINNER, M. D., Kansas City, Missouri

THE prolific use of the X-ray has forced surgical attention to the anatomical reduction of fractures. Anatomical reduction is a worthy ambition, but our appreciation of functional reduction has been obtunded thereby. I desire to show you that less attention to fragments need be displayed if the joint surfaces are in correct relation and the lines of weight-bearing force are functionally satisfactory. In an attempt to reduce to mathematical exactness the relation of certain weight-bearing surfaces, I present for your consideration certain studies upon two of the commonest joint injuries; i.e., fractures at the ankle and fractures at the wrist. My work upon the shoulder and hip is not as yet crystallized into simple estimations.

These studies have been pursued with the idea of bringing this method of prognosis within the reach of the physician outside of the larger cities and where the X-ray apparatus may be of small capacity. The mathematical diagrams herewith presented may be applied to any such negative if the directions regarding the position of the parts are followed.

FRACTURES AT THE ANKLE

Axiom.— The functional result of an ankle fracture depends upon the proper reduction of the astragalus so that the line of weight-bearing force which passes through the center of the tibia also passes through the astragalus at its center.

Directions for exposures of the ankle. The illustrations which serve to amplify this article are reductions from 5 x 7 inch plates. The X-ray exposures are always made with the leg resting upon the long surface of a right angle frame, and the foot is supported by an Esmarch bandage or heavy rubber bands against the right-angled foot piece. The X-ray tube is centered, in the adult, one inch above the center of a line connecting the external and internal malleolus. An exposure with even the crudest apparatus will give a nega-

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 1. Normal ankle-joint taken in the anteroposterior position and showing that a line drawn through the center of the tibial shaft will pass through the center of the astragalus. Note the normal apposition of the astragalus under the tibia.

Fig. 2. An old fracture of the ankle, with extreme displacement of the astragalus. Note the relation of the astragalus to the line through the tibia. The weight of the body in walking is carried to the foot by the shaft of the tibia, and if the astragalus is not centered under the tibia, there is more or less pain and discomfort in walking.

Fig. 3. An old fracture of the ankle. It will be noted that the ankle mortise is broader than normal, and the astragalus is not centered under the tibia as the space between the astragalus and the internal malleolus indicates. The line down the center of the tibia shows that there is more of the astragalus external to the line than in the normal ankle.

tive upon which the line of weight-bearing force may be charted.

A line drawn through the center of the shadow of the tibia extends through the center of the shadow of the astragalus. This line may be charted upon the anteroposterior X-ray negative by applying a line to the center of the tibial shaft shadow and extending it downward through the shadow of the astragalus. This vertical line should always extend through the center of the astragalus if the ankle joint is in normal position.

The charting of this described line of weight-bearing force may be made upon the finished negative by means of any straight edge, preferably a celluloid rule.

The interpretation of the plate depends not so much upon the shadow of the lines of fracture as upon the shadows of the relative position of the tibia and astragalus. One must judge of the injury by the position of the astragalus. The anteroposterior position negative gives us the information regarding the mortise between the astragalus and tibia.

To judge the normal or abnormal relationship of the tibio-astragaloid articulation, we must realize that the head of the astragalus is firmly mortised between the lower end of the fibula and the internal malleolus, and is directly beneath the broad articulating and weight-bearing surface of the tibia. This gives only sufficient room between the internal lateral border of the head of the astragalus and the tibia for some ligaments and the action of the joint. This space increases with the slightest torsion fracture of the fibular malleolus for it is due entirely to the integrity of the lower end of the fibula that the astragalus remains mortised correctly.

Provided we have established, by the X-ray, the diagnosis of a torsion fracture of the lower end of the fibula, we are at once interested in methods of reduction and fixation which will promote a functionating ankle as a result. Examination of

Fig. 4.

Fig. 5.

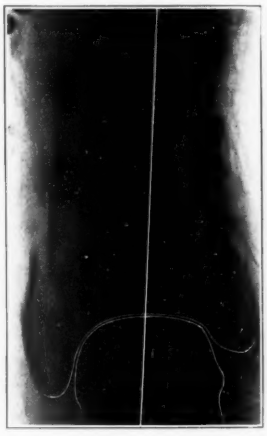


Fig. 6.

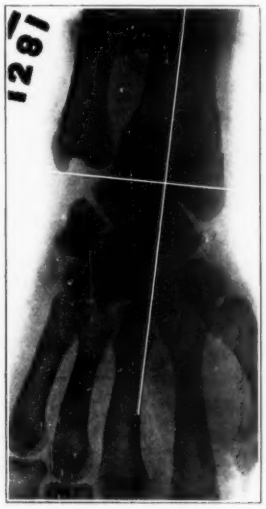


Fig. 7.

Fig. 4. Pott's fracture of the ankle. Taken after attempted reduction. More than three-fourths of the astragalus is external to the plotted line, indicating that further reduction is necessary.

Fig. 5. Fracture of the lower end of the fibula after reduction. It will be noted that the anatomical reduction of the fibula appears to be very good, but the position of the astragalus is not satisfactory, and if the ankle were left in this position there would be an abnormal line of weight-bearing force. This illustrates the necessity of interpreting X-ray negatives with regard to the functional prognosis rather than to the anatomical position of the fragments of a fracture.

Fig. 6. Fracture of the lower end of the fibula without a perfect anatomical reduction of the fragments but with a very good reduction of the astragalus. This case made a good recovery.

Fig. 7. Illustrating the plotting of lines upon the postero-anterior negative of the wrist. In the naturally extended hand the long axis of the radius usually passes through the interval between the second and third metacarpal bones, but this may be a trifle altered if the hand is cramped or influenced by bandages. Note that the styloid of the radius is entirely distal to the horizontal line, which is at right angles to the long axis of the radius and just touches the tip of the ulnar styloid.

Fig. 8.

Fig. 9.

Fig. 10.

Fig. 11.

Fig. 12.

Fig. 13.

Fig. 14.

Fig. 15.

For legends, see p. 241.

a large number of results of ankle fractures, both in hospital and private practice, has taught me that the proper treatment of these cases is extremely difficult. True, the fracture is a slight and simple affair, but the broad, painful ankle that so frequently results merits our attention to details.

The secret of the correction of this fracture lies in the proper adaptation of the astragalus to the tibia. The weight-bearing line of the tibia is transmitted to the foot by way of the articulating surfaces of the tibia and astragalus. It is essential that this relationship be maintained to insure a useful ankle-joint. For years we have enjoyed an epigram something like this: "Take care of the tibia, as it bears the weight of the body; the fibula has no function and will take care of itself." In contrast I would advocate: "Reduce the astragalus and avoid painful ankles." And to properly reduce the astragalus requires attention to the fibula, for by the lower end of the fibula alone is the astragalus properly adapted to the weight-bearing tibia.

Now we know that the integrity of the ankle depends upon how well the low end of the fibula performs this mortising function. The large majority of ankle fractures and chronic ankle sprains are fractures of the external malleolus. It frequently happens that the position of the fragment in a fracture of the external malleolus is in apparently good position, but the mortise of the ankle-joint is sprung open by the slight outward displacement of the fibular fragment. Conversely, we may have a poor alignment of the fracture but a splendid reduction of the astragalus. It is, therefore, unnecessary to pursue futile manipulations of the fibular fragment if the astragalus is in correct alignment.

One will readily see that the slightest displacement of the astragalus, externally, presents a widened ankle space, and if this is not corrected the patient will ever after have an abnormal line of weight-bearing force. In some cases the ankle may adapt itself to the new position, but a little attention to the position of the toes will avoid many painful and weak ankles of tardy convalescence. A good motto for the surgical attendant should be: "Watch the toes; turn the toes in."

It does not matter what the form of fracture at the ankle, whether simple or serious, for, if the anteroposterior X-ray negative shows the astragalus centered under the tibia, the prognosis of a functional result will be more favorable than any anatomical alignment of fragments.

FRACTURES AT THE WRIST

Axiom.— The entire styloid process of the lower end of the radius is constantly distal to a line which touches the tip of the ulnar styloid, which line is at a right angle to the longitudinal axis of the radius. The functional result of fractures of the lower end of the radius depends upon the reduction of the radial styloid to this position.

Method of X-ray exposure of the wrist. The difficulty in obtaining and interpreting lateral views of the wrist has led to this charting of the wrist-joint upon a single postero-anterior X-ray exposure. The affected wrist and hand are placed with the palmar surface upon a 5 x 7 inch X-ray plate with the one end of the plate at the web of the thumb. The tube is centered over a point midway between the styloid processes of the radius and ulna. The resulting plate provides shadows of the proximal portions of the metacarpals, the carpus and about three inches of the lower end of the radius and ulna.

Fig. 8. Fractured radius. Note that the styloid of the radius is proximal to the cross line, indicating a backward and upward displacement of the radial head. The long axis of the radius now passes down between the third and fourth metacarpal bones, owing to the rotation of the hand and carpal area with the displaced radial head.

Fig. 9. Lateral view of the same case as Fig. 8. This is shown to prove the interpretation made under Fig. 8, and to show the direction of the joint surface of the lower end of the radius when there is a backward and upward displacement. Normally the joint surface of the lower end of the radius exhibits the dorsal edge distal to the palmar edge, but when there is any displacement backward and upward it exhibits just the opposite, which is here illustrated. See Fig. 11 for normal position.

Fig. 10. Fractured lower end of the radius reduced. This is the same case as Fig. 8 after reduction, and while in plaster cast. Note that we now have the normal plotting of the two lines, and the styloid of the radius is distal to the cross line.

Fig. 11. Lateral view of Fig. 10, showing the head of the radius reduced and the joint surface of the radius in normal position. Compare this with Fig. 9. This is the normal position of the joint surface of the lower end of the radius with the dorsal edge distal to the palmar edge.

Fig. 12. Separation of the lower end of the radius at the epiphysis with backward and upward displacement. Note that the radial styloid is proximal to the cross line, which may be interpreted as indicating the displacement of the lower end of the radius.

Fig. 13. Lateral view of Fig. 12, which proves the interpretation of Fig. 12. Note the abnormal position of the joint surface of the lower end of the radius.

Fig. 14. Fracture of the lower end of the radius with displacement of the radial head backward and upward as indicated by the position of the radial styloid in relation to the cross line.

Fig. 15. Lateral view of Fig. 14, proving the interpretation and indicating the abnormal position of the joint surface of the lower end of the radius.

Anatomical considerations. In the antero-posterior radiograph of the normal wrist, a line drawn through the center of the long axis of the radial shaft shadow passes between the proximal epiphyses of the second and third metacarpal bones. In backward displacements of the fractured lower end of the radius the radial rotation of the carpal area and metacarpals usually bring this line between the third and fourth metacarpals. But this single line is not sufficient to judge the radial position from the single postero-anterior X-ray negative. So we must add the line at right angles to this longitudinal axis of the radius at a point corresponding to the tip of the ulnar styloid. If the styloid of the fractured lower end of the radius is behind this transverse line, it is not properly reduced, and if left in this position will remain a functionally imperfect wrist.

It does not matter what the nature of the fracture at the lower end of the radius, whether it be an epiphyseal separation in youth or a comminuted fracture of the aged, if the reduced lower end of the radius provides the above normal charting, one may be fairly sure of the functional result.

The functional result depends upon the proper adaptation of the carpal articulating surface of the radius. The articulating surface of the normal radius with the carpus is tilted so that the dorsal edge of the lower end of the radius is always distal to the palmar edge. The correct position of the radial styloid according to the above charting necessitates the normal anatomical position of this articulating surface.

Many writers have described the dropping of the ulna, or the separation of the ulna, as the cause of bad results in Colles's fracture. But these objective findings usually depend upon the tilting backward of the radial articulating surface at the wrist. Furthermore, it does not matter if the apposition of the fragments is faulty in contour if the extension is sufficient to replace the articulating surface to its normal weight-bearing position.

It is the tilting backward of the lower end of the radius that interferes with the function at the wrist, and this can only be overcome by the full extension of the fragment and the consequent normal position of the articulating surface of the radius.

This charting has been applied to X-ray negatives of all varieties of fractures at the lower end of the radius with satisfactory results. Do not try to straighten out the hand where the wrist is fractured, but let it assume a comfortable position. Any unnatural change in the position of the hand alters the relation of the metacarpals to the longitudinal axis of the radius, the first line of our calculations; but such a change in the metacarpal position does not in the least alter the plotting of the position of the radial styloid with relation to the second line, which is transverse to the first line and just touches the tip of the ulnar styloid.

If the entire styloid process of the lower end of the radius is distal to the transverse line at the ulnar styloid process, it implies that the articulating surface of the radius with the carpus is in a correct position for a good functional result.

Nature is wonderfully tolerant of fragments if she can maintain her functional joint surfaces.

CASE OF PERNICIOUS ANÆMIA TREATED BY SPLENECTOMY; MARKED IMPROVEMENT

By W. S. HARPOLE, M. D., and CHARLES M. FOX, M. D., Chicago

WE desire to report this case thus early because the results are sufficiently suggestive to warrant further operation in this fatal disease. The case was operated in ignorance of the five German cases that have been reported since August, 1913.

The reasons for operating were the following: (1) A case of pernicious anæmia steadily going from bad to worse under the usual medical treatment. (2) The presence of an easily palpable spleen which could not be accounted for by previous malaria or other disease. The enlargement of the spleen suggested the possibility of an hypertrophy due to overactivity of the organ, the possibility that the spleen was perniciously active in a function generally accredited to it, namely, the destruction of red cells. The case has improved since the operation, as have the five German cases, some of which are reported as completely healed. The phenomenon of a great increase of nucleated red cells after the removal of the spleen corresponds to the other cases reported. The color index has fallen ever since the operation.

The patient received a back-set sixty days after the splenectomy, by having an appendicitis with ruptured appendix and somewhat diffuse peritonitis, which was treated by appendectomy and drainage, from which he made a rather slow recovery. At present he is up and about, feeling fine and gaining in health and strength. The history is as follows:

J. D. B., aged 40, comes from Iowa; characteristic appearance for pernicious anæmia. Moderate spinal changes. Gives a history of loss of weight and strength for two years. Had lost much weight in the few months before coming to Chicago, because he had been given a vegetarian diet, to correct the possible auto-intoxication. The blood showed, July 14, 1913 (counted by the Columbus Laboratory): Hæmoglobin, 40 per cent; reds, 1,200,000; whites, 2800; poikilocytes, nucleated reds and megaloblasts. Color index, 1.6.

Treatment ordered: Sodium cacodylate, grs. 2, hypodermatically, every other day. Large doses of hydrochloric acid after meals. Full diet, with a large quantity of meat. The count on August 29, 1913: Hæmoglobin, 37 per cent; red cells, 1,113,600; whites, 5000. Index, 1.69.

The patient was operated upon September 8, 1913. He was given gas-ether. Incision through outer border of left rectus. Spleen delivered after breaking up a few adhesions to diaphragm. Gastro-enterostomy clamp was put on pedicle. The vessels of the pedicle were markedly atheromatous, so much so that the soft clamp ruptured one or two good-sized arteries. The pedicle was doubly ligated and the wound closed. The spleen weighed twice the normal; was firmer than normal. A number of small millet-sized nodules showed on the surface. The cut-surface showed a few of these same nodules scattered through the splenic pulp. Microscopically, there was an increase of connective-tissue elements and decrease in lymphoid structures; also some hyaline degeneration in the blood-vessels. The nodules already mentioned were of connective tissue. The blood-vessels of the spleen were increased in number. The diagnosis of the pathologist was chronic splenitis. Following the operation the patient ran a temperature of about 100° F. for three weeks. This gradually returned to normal. The patient's lemon-yellow color disappeared in ten days. The œdema of face and ankles left in two weeks and has not returned. The drowsiness that the patient had always experienced disappeared three days after the operation. The patient rapidly gained strength and got out of bed on the eighth day, walked about in two weeks, and left the hospital October 5th. The blood counts after operation were: September 10th, two days after operation: Reds, 960,000; hæmoglobin, 20 per cent; whites, 22,000; index, 1.4. September 14th: Reds, 1,100,000; hæmoglobin, 25 per cent; whites, 18,000. September 18th: Reds, 1,212,000; hæmoglobin, 25 per cent; whites, 14,000. September 22d: Reds, 1,420,000; hæmoglobin, 30 per cent; whites, 14,000. September 28th: Reds, 1,618,000; hæmoglobin, 35 per cent; whites, 11,000. October 3d: Reds, 1,760,000; hæmoglobin, 40 per cent; whites, 9400; index, 1.07. November 6th: Reds, 1,968,000; hæmoglobin, 45 per cent; whites, 18,000.

On November 7th the patient was operated for appendicitis, whose presence was possibly indicated by the high leucocyte count of the day before, although he did not complain of a symptom of it until on his way home from the laboratory where the count was made. After the operation for appendicitis the blood rapidly fell off, but soon began to recover. The counts are: November 9th: Reds, 1,440,000; whites, 18,000. November 11th: Reds, 1,220,000. November 27th: Reds, 1,412,000. December 18th: Reds, 1,620,000; whites, 12,000; hæmoglobin, 30 per cent.

Ever since the splenectomy the blood has shown a great number of normoblasts, many more than before the operation. The patient has gone on a trip to the southern part of Illinois, feeling fine, gaining strength rapidly, having a color not noticeably different from the normal.

THE EXTRAPERITONEAL CÆSAREAN SECTION

Its Place in Obstetrics

By WILLIAM R. NICHOLSON, A. B., M. D., Philadelphia

IT is an interesting fact that the operation known to-day as the extraperitoneal supra-symphyseal cæsarean section originated in the minds of practical obstetricians more than one hundred years ago. The first man to propose the procedure was Jorg, who in 1806 advocated an intervention in impossible labor which was based upon the observations of many accoucheurs, among whom Boer of Vienna is mentioned, that after the rupture of the upper part of the vagina and lower uterine segment, the fœtus may be found in the abdominal cavity. At that time the classical operation was practically fatal; Blundell, in his "Principles and Practice of Obstetrics," published in 1834, gives a list of twenty-three cases, the only ones performed in England before the year 1821, with fatal results in all except one. This successful case was operated by Barlow in 1793. As late as the year 1866, Playfair stated before the Obstetrical Society of London that the mortality was 89 per cent, and in 1882 Greenhalgh, of Saint Bartholomew's Hospital, could only report one successful case among the ten operated by him.

From the earliest times of which we have record it had been understood that entrance to the peritoneal cavity was accompanied by the most serious risks, and though the real reason was not understood, the operators were more or less close to the truth. Thus Jorg, in his text-book published in 1820, says that the classical operation is fatal in 95 per cent of all cases because of the entrance of air into the peritoneal cavity. In 1860 Nelaton could find no successful case operated in Paris within sixty years, and it is a fact that from 1787 there was no successful case in that city until the year 1879, when Tarnier performed his first Porro operation. The conditions in Vienna during this period were identical, according to Spath, who states that up to the year 1877 there is no record of a successful case in that city. Before 1882 from 64 per cent to 85 per cent died in the best German clinics. It is hardly to be wondered that obstetricians came to the conclusion that not only must the attempt be made to decrease this high mortality, but that the only method was by the avoidance of the peritoneal cavity. One of the men thus impressed was Ritgen, who refers to Mothe of Paris as advocating a procedure practically identical with the Duhrssen vaginal section. In 1821 Ritgen performed the Jorg operation in a slightly modified form but with a fatal result, encountering such severe bleeding that he was forced to complete the operation by the classical method.

It is a pleasure to find that only a few years later eminent American physicians were also concerned with this same question. In the year 1824 Physick proposed to Dewees that the approach to the uterus be made extraperitoneal. This advice is quoted in the text-book of the latter, published in Philadelphia in 1826, and was founded upon anatomical studies of Drs. Horner and Physick. While this operation was never performed, it is recognized by Latzko as being practically identical with the technique known to-day by Sellheim's name. Shortly before this time Baudelocque the younger suggested gastro-elytrotomy. He made two attempts to follow the technique of Ritgen, in the first instance being compelled to desist because of severe vaginal bleeding, while in the second he injured the external iliac and was forced to tie the common iliac artery.

From this time there seems to have been a cessation of activity regarding the matter until the year 1870, when Thomas performed the operation of laparo-elytrotomy. His method was as follows: manual dilatation of the cervix followed by an incision from the symphysis to the anterior superior spine of the ilium. He then dissected down to the peritoneum, which was separated and pushed upward. The cervix and vaginal wall were then exposed and the latter incised upon a sound introduced into the vagina as a guide. The cervix was now drawn through the wound in the vaginal wall into the external wound in the abdominal wall and the child delivered. By this method Thomas performed two operations, the first upon an eclamptic with a fatal result, while the second, in the case of a young primipara with contracted pelvis, was a success. In 1876 Skene had the pleasure of saving two women by this method, and in 1878 the American Gynecological Society sanctioned the operation and gave it the name laparo-elytrotomy. Up to the present time there have

been fourteen of these operations reported: two each by Thomas, Duncan, and Jewett, while Skene had five and Gillette, Hime and Edis each one. The mortality of these cases was 50 per cent for the mothers and 42 per cent for the children, and it is to be remembered that the histories show the majority to have been in so serious a condition that they would have been very bad risks under any plan of treatment. The bladder was wounded in six instances, and the risk of this accident and the general technical difficulty of the procedure caused a halt in its performance, together with the fact that the introduction of asepsis rendered it an unjustifiable operation. It may be stated, however, that in competent hands, at the present day, it should not have a greater mortality than the recently devised Duhrssen-Solms technique.

In preparation for this study I sent out a number of letters to well-known operators in the United States, asking for their experience with and their opinion concerning the present-day extraperitoneal cæsarean, and among the letters in reply I received one from Dr. G. K. Dickinson of Jersey City, which was of so much interest to me that I incorporate it here, since it adds another unpublished case to the list of laparo-elytrotomies and gives a most vivid picture of the obstetrical situation of thirty years ago.

"In reply to your letter of yesterday, I would say that the only operation of that kind with which I was associated occurred so long ago that the notes are meagre and my recollection, perhaps, not altogether correct, although very vivid. Dr. J. J. VanVorst and myself were the surgeons. Dr. W. F. Watson and some others were assistants. The neighbors were aroused, and vengeance was to be meted out to us if we attempted anything. The woman was not consulted. Her husband was tentatively agreeable to have something done. Her history was of several confinements, craniotomies, an agonizing time, and no result. Dr. VanVorst and myself feared the outcome of this case and were very much impressed by an article which appeared at that time termed laparo-elytrotomy. I can remember that we congregated in Dr. VanVorst's office; then, when a messenger was sent, we went around the corner and stood behind the trees with our satchels awaiting the signal. The signal being given we went into the house. It was one of those dark dirty tenements and the last place in the world to do this kind of a job. The woman had been in labor several days. The pelvis was narrow; cervix well dilated and soft; secretions were abundant and the pains came on at five-minute intervals. She was considerably exhausted from a long period of ineffective labor. We put her to sleep, washed her up, used the carbolic of the day, and made an incision on the right side above Poupart's ligament, cutting through all the structures. Coming down on the peritoneum we dissected it up until the vagina was reached. This was opened and the baby extracted. The baby died, also the mother. This was done in a little room about twelve feet square, with one window. The curtain had to be drawn on account of fear of the public. If I remember correctly the woman lived through the following night, but succumbed the next morning from exhaustion, perhaps beginning sepsis. I remember there was some bleeding but not very abundant. What there was we easily controlled by packing. The difficulty was in stripping up the peritoneum and reaching the vagina. Surgery in those days being novelties to the most accomplished, and particularly to us, the local conditions and the psychology of it all rendered the operation more than usually disturbing. So far as I can bring my memory back there is no other incident of importance connected with this case."

Between the years 1891 and 1896 the mortality of the classical cæsarean section may be estimated as about 38 per cent; in the year 1902 it had fallen to 20 per cent and in 1904 it reached the low-water mark, 12 per cent. This wonderful reduction in mortality was, of course, in greatest measure due to the improvement in technique rendered possible by the introduction of asepsis, but it is always to be remembered that it was also to some extent the result of a better selection of cases, it having been early appreciated that the seriously infected parturient was no fit subject for the procedure. As time went on and better statistics became available, it was still more definitely recognized that unless something more was to be added to the equipment of the obstetrician the operation of craniotomy must still be called upon in not infrequent cases, since the mortality of 8 per cent in general practice for this operation is far better than a percentage of 34.3 per cent, which can be shown by statistics to be about what may be expected in a series of cases subjected to the classical operation, after frequent attempts at delivery or repeated examinations have been made by unskilled attendants. The only other alternatives were the much vaunted pubiotomy, hysterectomy either with the extraperitoneal treatment of the stump, or else the so-called cæsarean hysterectomy, and no one of these

operations satisfied the requirements, since pubiotomy was found to be a dangerous procedure in neglected cases from the standpoint of maternal sepsis, while the child had to undergo the ordeal of pelvic delivery at a time when, weakened by the previous traumatisms of labor or human endeavor, it was illy conditioned to withstand any added insults. The removal of the uterus, although giving a better percentage of maternal results than the classical operation in the presence of severe sepsis, could not be considered ideal, if for no other reason than the destruction of the power of reproduction. In fact it was felt that it was a wiser procedure to perforate, and by thus destroying a child, often beyond resuscitation, to save the life of the mother for future childbearing.

Such were the reasons for the proposition, first made by Frank of Cologne in 1907, to perform the operation of cæsarean section extraperitoneally and above the pelvic brim. That there was a real demand for some improvement which would enable the profession to cope with the seriously infected case is evidenced by the enthusiastic diligence of Frank's German colleagues in perfecting and modifying the original technique. There are in the neighborhood of twenty operations now on record for the attainment of the suprasymphyseal extraperitoneal delivery. While in many of them the differences may seem unimportant, to the investigator far removed from the sounds of battle it is necessary, for the full understanding of the subject, that a short résumé should be incorporated here, though among the number there are certain performances which can only appeal to the surgical contortionist.

The anatomical facts upon which all these operations are dependent are, first and most important, the distention of the lower uterine segment; secondly, the migration of the peritoneum during pregnancy, whereby the bladder is more or less deprived of peritoneum and becomes in late labor uncovered by the peritoneum above and in front; thirdly, the growth of the peritoneum due to the hypertrophy of pregnancy; and, finally, the loosening of the peritoneal reflection over the lower uterine segment, which, as is so well shown in vaginal cæsarean operations, enables it to be separated from the uterine wall with the greatest ease.

As early as the year 1908, Holzapfel was able to enumerate sixteen different methods for the performance of the suprasymphyseal extraperitoneal cæsarean section and since that time there have been several further modifications, but these, with the exception of the Duhrssen-Solms technique, are of little importance. The last mentioned operation is unique and will be mentioned later.

Frank of Cologne was the first man in recent times to propose the extraperitoneal method, and therefore the classification of these various procedures is headed by his method, which was as follows: The abdominal wall was opened by a Pfannenstiel incision, and the parietal peritoneum was incised transversely, as was also the uterine; the latter was then separated from the anterior face of the uterus and joined by stitching to the parietal peritoneal leaflet; finally the uterus was incised transversely. This method is known as Frank's first technique and is in reality a transperitoneal operation. His second method attempts to fulfill the aim of true extraperitoneal attack and was performed as follows: After incision of the abdominal wall as in the former method, a small opening is made in the parietal peritoneum for the orientation of the bladder, and this organ is freed from its peritoneal coat by blunt dissection, or, where needful, by a few cuts of the scissors; the incision into the parietal peritoneum is then closed and the uterus is opened as in the former method.

The Veit-Fromme original technique is identical with Frank's first method except that after the uterus has been closed the peritoneal flaps are again separated from one another and united as before operation. As a modification of this technique the same authors have presented the following, which is in our opinion the best of all the manifold methods by which this operation may be performed: the abdominal wall is incised longitudinally, as is also the peritoneum, both parietal and visceral; these two layers of peritoneum are then brought into apposition by clamps, and after the delivery has been accomplished through a median longitudinal incision into the lower segment, the peritoneal flaps are reunited in their original relations.

One of the earliest to enthusiastically adopt the idea of Frank and Veit was Sellheim, and he has published four different modes of procedure. His first method is a true extraperitoneal technique: Pfannenstiel incision through the skin and fascia, separation of the recti longitudinally, separation of the subperitoneal tissue from the under surface of the recti, distention of the bladder with solution to aid in its orientation, loosening of the peritoneum from the top of the bladder, and finally the upward displacement of the unopened sac of peritoneum with coincident downward displacement of the bladder below the symphysis.

The uterine incision is made in the middle line of the lower segment and is vertical. In his second method the technique is as just detailed until the parietal peritoneum is reached; this is incised transversely and through this incision the uterine peritoneum is similarly incised and the two flaps thus formed are united by stitches. The peritoneum is then pushed up and the bladder is displaced downward. The only difference between the method just described and his third method is that in the latter the folds of peritoneum are united by a double layer of catgut stitches. Sellheim's fourth method, however, is a wide departure from his previous three plans and is an attempt to treat all cases, of whatever grade of infective virulence, by suprasymphyseal operation. It is known as the method by utero-abdominal fistula and is performed as follows: Median incision through all abdominal layers, including the serosa of the lower segment. The parietal serosa is then stitched on the skin surface and the uterine serosa, after lateral separation from the uterine wall, is stitched upon the parietal near the edge of the skin incision. A median incision is then made in the lower segment of the uterus, and the incised edges are drawn forward into the abdominal wound during the delivery. After the birth has been completed the edges of the uterine wound are sutured to the skin surface. The orifice thus left is packed and allowed to heal by granulation.

Pfannenstiel advocates the incision known by his name, through the skin and fascia with a longitudinal separation of the muscles, a longitudinal incision in the peritoneum, and a longitudinal incision in the lower segment of the uterus.

Baumm advises the same technique as Veit's first method, with the exception that he sees advantages in the clamping of the peritoneum, instead of stitching it, during the delivery.

Rubeska offers a method for badly infected cases. He makes a median incision in the abdominal wall and stitches the parietal serosa around the area to be incised before opening the uterus. The uterine wound is treated by the open method. This is a similar procedure to Sellheim's fourth method.

Doederlein is a strong advocate of the true extraperitoneal technique and attempts it by an initial inguinal incision followed by dissection of the parametrial tissues without exposing the peritoneum. After reaching the lateral aspect of the lower segment, the bladder is displaced laterally without separating the peritoneum from it, and the uterus is opened in the median line. Drainage is a part of his usual technique.

Czyzewicz prefers the median abdominal incision until the peritoneum is reached and then follows Sellhem's first method for the balance of the procedure.

Hoffmeier in his earlier work followed the technique of Veit's second method, the only difference being a continuous seroserous suture, non-separation of the united flaps of peritoneum and the use of drainage. His second method simply consists in a median section through the abdominal wall and through the uterus as low as possible. One of the methods which seems to have attracted a large number of foreign operators is Latzko's. This operator fills the bladder with 150 ccm. of sterile solution and then makes a 12 cm. incision through the recti, which are laterally displaced. The bladder is then loosened from the lateral aspect of the cervix to a degree which permits a side displacemnt of the former with one rectus muscle; the uterus is opened longitudinally and the uterine wound is drained.

Probably the most remarkable form of intervention is the procedure known as the Duhrssen-Solms technique. In this method the abdomen is opened by an incision parallel to Poupart's ligament and the tissues are separated by blunt dissection until the uterus is reached; during this dissection the epigastric vessels must be ligated. A vaginal cæsarean is then performed and the wound in the uterus and the wound in the abdomen are brought into coincidence by traction, the child being delivered above the symphysis.

A decided simplification of the above confusing array is made possible by classifying them on the basis of essential differences; thus the Frank, Veit-Fromme, Baumm, Pfannenstiel, Hoffmeier, and the second method of Sellheim are all transperitoneal methods, their differences only consisting in various means of closing the peritoneal folds (parietal and visceral) and whether or not these same folds of peritoneum, which have been united for the moment, are again restored to their original anatomical relations before closing the abdomen. There are three other divisions by which all the other important methods may be classified, as follows: the utero-abdominal fistula technique of Sellheim and Rubeskathe, true extraperitoneal technique of Sellheim and Latzko, and finally the Duhrssen-Solms technique. While opinions among the continental operators differ widely as to the value of the various operative methods described, and while, of course, each creator of an individual method is enthusiastically in favor of his modification, it seems fairly clear to a non-prejudiced investigator that the Veit-Fromme modification of the Frank operation

and the second Sellheim technique are the only methods which are truly surgical. Most of the other procedures, if not absolutely all of them, are either founded upon the principle of making the operation as difficult as possible, or else the distinctive modification is of such a nature that suspicion is aroused that the author was anxious to drive a nail upon which he might hang his name.

Sellheim makes the objection to Latzko's method by lateral undermining that it is not suitable for all cases, and to Doederlein's that sufficient space is often not afforded for an ample uterine incision, and that there may be, therefore, severe uterine lacerations produced by extension during extraction of the child.

In order that a definite foundation be established for the further consideration of the subject, the Veit-Fromme method and the second method of Sellheim will now be considered in detail.

Veit was one of the first to advocate the operation which is known as that of Frank. Fromme in 1908 reported eight cases from Veit's clinic, in the first four of which the technique was exactly that of Frank. In the fifth case the parietal and visceral peritoneum were separated after the birth of the child, and were restored to their normal relations. In the remainder of the series the transverse section through the skin, subjacent tissues and uterus was given up and in its place was substituted the longitudinal incision. This modification is performed as follows: the skin, fascia and recti muscles are separated vertically and a vertical incision is made into the parietal peritoneum. The visceral or uterine peritoneum is then incised vertically in the median line of the lower segment, beginning at the lowest possible point of the vesico-uterine plica and extending upward to the point at which the peritoneum becomes intimately attached to the uterine wall. In advanced labor the lower segment has become so distended that a space of from 10 to 12 cm. is available for this incision. The peritoneum of the visceral layer is then mobilized somewhat laterally, which is easily done by blunt dissection, and then the two peritoneal flaps, visceral and parietal, are either stitched or clamped. A vertical uterine incision is made into the lower segment and the child is delivered. The placenta may be delivered at once, as in the classical operation, or it may be awaited and expressed by fundal pressure either before or after the uterine incision is closed. The uterus is best closed by a running stitch, reinforced by a second one as in intestinal suture. The abdominal wound is closed by the layer method and no drainage is used. The chief advantage of this method, in the opinion of its originator, is that the bladder is not disturbed. (There is one distinct disadvantage in this technique, however, in that there may be difficulty in obtaining ample space for the uterine incision if performed during the early stages of labor. It is well to remember also that the fear of bladder necrosis from separation is in all probability not well founded.)

In Sellheim's original operative procedure, he made the attempt to separate the bladder from the peritoneal fold without opening the latter, and then by upward displacement of the unopened peritoneum and downward displacement of the bladder to obtain enough space to deliver the child. It was soon found, however, that not only was it impossible, in many cases, to avoid opening the peritoneum, but that the bladder was liable to injury by the attempts at separating the peritoneum unopened. This method has, therefore, been very generally given up, and he has substituted the following, which is in reality not a true extraperitoneal but rather should be designated as a transperitoneal method. He operates under scopolamine-morphine twilight sleep, with spinal anæsthesia. The high Trendelenburg position is used, and at present the incision is made in the longitudinal direction instead of the Pfannenstiel, which was the case in his earlier works. The fascia and recti muscles are separated vertically and the peritoneum is separated from the under surface of the recti to a considerable distance on each side along the upper border of the empty bladder. The peritoneum is now opened at the attachment of the urachus to the bladder. The incision in the peritoneum is prolonged to left and right along the firm attachment of the peritoneum to the bladder, curving the incision laterally and posteriorly on both sides toward the floor of the vesico-uterine fold, while the bladder is drawn forcibly forward. A transverse incision is now made at the boundary between the loosely attached peritoneum in the vesico-uterine fold and the firmly attached bladder peritoneum. This transverse incision is prolonged on both sides into those portions of the anterior delimiting incisions which curve toward the side and back. The delimiting incision begins at the portion of the bladder peritoneum which remains undisturbed. The visceral layer of the peritoneum is now dissected away from the uterus in an upward direction as far as possible. The two leaves of peritoneum, the parietal and the visceral, are now united by suture of their free edges, and care must be exercised here to avoid incomplete closure of either extremity of

the transverse incisions; in other words, it is necessary to completely close the peritoneal cavity by suturing the peritoneal leaflets well out onto the broad ligaments if necessary, not only to avoid contaminating the peritoneum but also to avoid the possibility of omental or intestinal strangulation. The bladder is now separated from the cervix and drawn forward over the symphysis and pressed against it. (It is better to allow the bladder to drop down behind the symphysis after its separation from the cervix and to retain it there by a suitable retractor.) The anterior face of the lower segment being thus prepared, the incision is made in the median line in a vertical direction. Primarily but about 5 cm. are incised, beginning at a point about 4 cm. below the upward displaced peritoneal fold. The tip of the special lever, devised by the author for the delivery of the head, is introduced into this slit by the aid of lateral stretching and adapted to the head, while at the same time the lower angle of the wound, which is constantly stretching and becoming more accessible, is incised as far as is necessary to allow the full application of the instrument. The head is now raised from its bed and pried forward, and one pole of the presenting part is seized. The artificial cervix (uterine wound) is continued downward as far as needful for the passage of the entire head. The child is delivered, and the initial sutures passed at the upper and lower angles of the uterine wound. The peritoneal suture line is now carefully inspected and then the placenta is delivered manually and a uterine tamponade inserted, its lower end being led into the vagina. The uterine wound is closed by interrupted sutures, and this suture line is reinforced by a continuous catgut stitch, with the exception of a small space at the lower angle of the wound, which is left open in order to have a safety valve through which one may later reopen from the vagina, if there be need.

If at the time of delivery there be infection present, a strip of gauze may be carried through the lower angle of the wound into the vagina and the abdominal wound be drained by a Mikulicz. If the case be a clean one the peritoneal reflection may be attached to the bladder and secured with a few interrupted sutures to the lower portion of the rectus muscle. The abdominal wound is closed by continuous suture of catgut in the muscles and fascia, and any skin method which is desired.

In this multiplicity of operative technique there is, as has been said, one salient difference which divides them all into two great groups, namely: whether or not the general peritoneal cavity is opened in the preparation of the lower segment for incision, or, in other words, whether the operation is performed in a truly extraperitoneal manner by lateral displacement of the bladder, vesical plica, and unopened peritoneal sac, or whether, in the performance, the abdominal cavity is temporarily opened. It goes without saying that, technically, extraperitoneal section means extraperitoneal section, a procedure carried out from start to finish without entering the true cavity of the belly, and this would be the ideal method but for two facts which render its use less favorable: first, that it is a much more difficult procedure than the transperitoneal method, and, second, that it does not offer any greater advantage in the way of improved results than does the latter.

In the consideration of the two methods of operating through the lower segment, it must be apparent to any trained abdominal surgeon that the attempt to proceed truly extraperitoneally will be vastly the more difficult, since, to obtain the best exposure, a lateral abdominal incision of quite considerable extent is primarily necessary, with subsequent blunt dissection and displacement laterally of the bladder and vesical plica, in order to make the only proper median uterine incision. It is hardly necessary to offer a detailed censure of the transverse or the lateral uterine incision, as the unnecessary dangers which they make imminent must be at once apparent. Moreover, in the true extraperitoneal method the peritoneum is actually opened in a non-negligible percentage of cases, even in expert hands, and there is also the danger of deprivation of the vitality of the peritoneum by its separation. In addition, the bladder is so likely to be injured that the advice is given by some to open the parietal peritoneal layer for its orientation, which is, of course, a confession of weakness on the part of the adherents of this method. In the consideration of this problem, then, it is to be understood that the term extraperitoneal cæsarean section is taken simply to mean that the selected portion of the lower uterine segment has been made extraperitoneal before its incision.

With these qualifying statements in mind we may now consider the first of the two major questions of the problem, namely, are we in possession of a method whereby infected cases may be treated with a favorable prognosis for the mother and the child? Academically this question may be answered as follows: Cases which present themselves with the signs of a developed infection of a severe grade are not suitable for this procedure, as experience has

shown that less hope is offered for the maternal life than is the case when other well-recognized procedures are employed; cases after hours of labor, with ruptured membranes, multiple examinations or attempts at delivery, perhaps with foul discharge, but without temperature, will show a better percentage of recovery than by the classical operation, but a certain proportion will be lost who could be saved by craniotomy or hysterectomy; cases long in labor, with ruptured membranes but without evidence of infection (the so-called "neglected case" of the German writers), will be more safely operated by this than by the classical procedure. The lack of the possibility of definite determinative classification so apparent in this academic answer becomes vastly more oppressive when one is confronted with the actual case in practice, since in reality the true decision as to the proper procedure must depend upon the type and virulence of the infection, a matter which cannot at present be determined. One case entering the hospital after some hours of labor but without temperature or other danger signal will develop fatal sepsis after delivery, no matter whether it be by craniotomy, pubiotomy, classical cæsarean, or the extraperitoneal cæsarean section, while another case identical in history and general symptomatology will make a good convalescence after any one of these operations.

In the early days of the development of this operation it was hoped that it had solved the problem by which it had been created, namely, the treatment of severely infected women in labor; and in looking over the literature of the past five years it will be noticed that the idea of utilizing this procedure in the delivery of clean cases was not generally thought of until its failure to meet the indications in the severe grades of infection had been demonstrated. That it will not save the virulently infected case is now admitted by at least the great majority of experienced German and French operators, and the most ardent supporters of this technique, as Frank and Sellheim, are now opposed to its use in the severe types of infection, so often imposed upon a long-suffering hospital service, after the outside attendants have exhausted themselves physically by illy conceived and often barbarously executed attempts at delivery. Indeed, in one of his first communications Frank denied the statement which was attributed to him, that by this operation a great majority of infected women could be saved, by definitely stating that women may be expected to die after this operation as well as after any other obstetrical procedure. In other words, it is to a great extent the type of infection which determines the subsequent history of the case, and for this reason it may be permissible to reiterate that the large amount of space which has been consumed in the last few years in discussing the relative merits of the true extraperitoneal and the transperitoneal procedure has been wasted, since the one, in a virulently infected case, is just as useless as the other to check the advance. Indeed, in the great majority of cases in which death has followed the true extraperitoneal operation it has been by peritonitis, and it is thus emphasized that in order to have a peritonitis develop it is not necessary to mechanically infect the peritoneal cavity, and that simple stitching cannot make a germ-proof wall against invasion. Moreover the attempt to perform the true extraperitoneal operation is often rendered useless by unobserved laceration of the peritoneum, while by interference with its vitality by deprivation of blood supply, the result of its separation, a lessened resistance to infection may result. In addition it is always to be remembered that infection, by virulent organisms, of the cellular tissue of the pelvis or of the myometrium may be the cause of fatal sepsis, either with or without the development of peritonitis. For these cases of virulent infection we can, therefore, claim no increased armamentarium.

In this attitude Sellheim, Frank, Pestalozza of Rome, Brandt of Christiania, Bumm, Doederlein, and Baisch are in accord. In this type of case, craniotomy on the living but probably potentially dead child or the operation of hysterectomy, either supravaginal, with the extraperitoneal treatment of the stump, or the cæsarean hysterectomy are still the only hope. It is to be remarked in passing that in some of these cases Sellheim is an advocate of the utero-abdominal fistula operation, but he has lately become more conservative in this matter and would at present advocate it only if there was an absolute demand for a living child and if the parents were willing to take the increased risk. Sellheim indeed divides these cases into three groups: first, cases suitable for the extraperitoneal cæsarean section; second, cases which demand craniotomy, and, third, fatally infected cases. He admits that this differentiation is not always possible and that the extraperitoneal cæsarean section is therefore still an experiment in some cases.

There are still two great groups of cases to be considered relative to their suitableness for this operation: first, true clean cases, in which it comes into competition with the classical cæsarean, pubiotomy, and vaginal cæsarean, and,

second, the large group of cases in whom infection may be suspected but in whom at the time of examination there is no infection demonstrable. These latter cases comprise the "neglected cases" of the German authors, and for them have a definite significance in that, by the use of this term, they designate a patient who has been long in labor, with ruptured membranes, and who has had multiple examinations and possibly attempts at delivery outside the hospital, but without temperature or evidently infected liquor amnii. It must be stated that some authorities, as Schauta, an enthusiastic advocate of the classical operation, would not include such cases as suspected or neglected cases, but as true septic cases only suitable for craniotomy.

It is to be remembered, then, that in a good many cases this distinction between the evidently infected and the possibly infected or neglected case does not work out practically. I have had a recent case which illustrated this to an unfortunate perfection.

A woman admitted to my service at the Presbyterian Hospital, without temperature or foul lochia, was subjected to cranitomy, as her baby was dead. The cord was prolapsed and pulseless, and her doctor stated that he had made a vain attempt to deliver her at her home by the use of forceps. On her admission it was evident that not much attention had been paid to the amount of cervical dilatation which should precede a forceps operation. Her condition of shock seemed to preclude an abdominal operation and so the craniotomy was done. She died in three days of septic peritonitis.

In other words, she was a case which was in all probability doomed after the first application of the forceps, for while it may be an interesting theoretical question whether she would have been saved by a rapid hysterectomy, I am of the opinion that no one of experience, in the absence of certain knowledge of the virulence of her infection, would have dared to have thus delivered her. In a word, then, until we are in a position to determine the presence or absence of infection by scientific tests, such as bacteriological examinations, we shall be unable to accurately determine the proper method of delivery in all cases which we are called upon to treat, and if we were to be placed in the position to make these determinative tests to-morrow, we should still be no nearer saving the virulently infected case than we are to-day. These cases will never be saved until the medical profession is so trained that they do not occur. When the middle class patient can obtain in her own home the prophylactic oversight before labor which our hospitals now afford to the free patients in their dispensaries, then and not till then shall we be able to obtain the results in our major operative obstetrics which are our everyday experience in abdominal surgery.

Keeping this fact in mind, that the differentiation between the virulent infection and the suspected or doubtful case cannot always be made in practice, the question presents itself as to whether we are able to handle mildly infected cases of labor demanding operative delivery more safely by the extraperitoneal cæsarean than by the classical operation. In answering or attempting to answer this question it is necessary to say that any opinion now expressed must be merely tentative, since there is as yet not enough evidence to satisfy an observer unprejudiced by enthusiasm.

There is, however, enough evidence to at least give us hope that a real advance has been made if, in a given case, it may be decided that it is simply necessary to protect the peritoneum from the direct contamination of the liquor amnii and the necessarily infected uterine wound.

If the statistics of Sellheim are examined we find that in 1911 he was able to report a series of forty-six cases, comprising twenty-one cases of contracted pelvis, fifteen of placenta prævia (all of which were clean), four of eclampsia, two of vitium cordis (one of whom had marked struma), one case complicated with a foul ulceration of the portio, one case of marked prolapse with pus decubitus, one case in which the dystocia was due to a ventrofixation, and one case of purulent meningitis, operated in the agonal period. Of these women five died, two of eclampsia, one of placenta prævia (anæmia), one from sepsis, and finally the case of purulent meningitis. The fœtal mortality comprised six cases; one each of the eclamptic and prævial placental cases, one of septic pneumonia, one due to pneumococcus infection in utero, one as a result of strangulation of the umbilical cord by a knot, and one of lues congenita (14 days post-partum). The child dying as the result of the maternal eclampsia was underdeveloped, and it is certainly fair to exclude the death due to a knot in the cord as well as the death the result of syphilis from the fœtal mortality for the purpose of this study, and to estimate the mortality of the children at three instead of six cases. Excluding the fifteen cases of placenta prævia, which were all clean, it seems probable that the results here reported are somewhat better on the maternal side than if they had been delivered by any other single method. Baumm reports a series of forty operations in two years. He had two deaths from peritonitis. Ten of these cases should be left out of consideration, as they had unruptured

membranes at the time of operation. Twenty-four were operated by the transperitoneal method and both deaths occurred in this group. With the exception of the ten cases all were of the suspicious class or, as he says, "more or less unclean." He is an enthusiast and believes that craniotomy, pubiotomy, the classical cæsarean, and to some extent premature labor, will be crowded out by this operation. He followed Latzko's technique and believes it to be the best.

Kustner reports fifty-five cases with two deaths from peritonitis. One of these was clean and one was suspicious. In neither fatal case was the operation truly extraperitoneal. All the other cases did well. Some of them had temperature in the puerperal period and two of the children were lost. He would limit the classical cæsarean to cases in which some other intra-abdominal operation is necessary. He would perform the extraperitoneal cæsarean section if, the membranes being unruptured, it is certain that the child cannot pass the pelvis. If there is some doubt as to the possibility of engagement he advocates the test of labor, with the performance of the extraperitoneal operation if it becomes necessary. Kustner would limit pubiotomy, even in multiparæ, and believes in the extraperitoneal operation in infected cases, and would employ it even if there was some question as to the condition of the child.

Doederlein advocates pubiotomy to 7 cm. conj. vera in multiparæ, as he considers the extraperitoneal operation to be more formidable. In infected cases both the extraperitoneal operation and pubiotomy invite danger, and craniotomy is indicated. In thirty-two cases this operator lost three mothers and two children and two other children died in the first week.

Baisch reports fifty cases. In three the true extraperitoneal type of operation was not possible and in sixteen there were small peritoneal wounds made by the separation. Three women died from eclampsia, ileus, and sepsis, respectively, and six of the children were dead at birth. This author thinks that the operation is best for the suspected case without temperature, but with a probable infection of the liquor amnii.

Henkel reports twenty-five cases and states that most of them were at least suspicious. He operated by the transperitoneal route entirely and had no maternal fatality. Opitz also operates transperitoneally. He is able to report a series of sixteen cases, of which some were suspicious, with one maternal death. Bumm lost three women in his series of forty-two cases, and the deaths were all due to infection. Walcher is an adherent of the transperitoneal technique. In his series of fifteen cases he lost one mother and two children. Romer draws the conclusion from his statistics that the mother is safer under the operation of pubiotomy, when performed in proper cases, than under the extraperitoneal section, but that the mortality of the children is greater. Thus pubiotomy gave, in his hands, a maternal mortality of 1.7 per cent and a fœtal mortality of 6.8 per cent, while the extraperitoneal section resulted in a 6 per cent maternal and but 2.4 per cent fœtal mortality. Latzko studied one hundred and fifty cases from the literature and calculated a general mortality of 7.3 per cent and a mortality from septic causes alone of 5.4 per cent. Jeannin collected one hundred and forty-eight cases with eight maternal deaths from infection, two from eclampsia and one from other causes, thus giving a maternal mortality of 7.45 per cent, with a purely septic mortality of 3.04 per cent. His statistics combined with Latzko's give a maternal mortality in one hundred and ninety cases of 7.37 per cent, and from infection alone a maternal mortality of 4.21 per cent. The morbidity was also high in this series, being 30.7 per cent total, with a morbidity of 25 per cent due to infection alone.

Lewis collected one hundred and two cases from the literature and calculated the maternal mortality to be 8.8 per cent while the fœtal was 8.6 per cent.

Taking the statistics which have been given as a basis for judgment, it appears then that we may admit that in the "suspected case," with the baby in good condition, we will conserve the interests of the mother by the performance of the suprasymphyseal extraperitoneal cæsarean section in preference to the classical, and that the mortality of the child from the operation should be but little greater than by the latter.

Now as to the employment of the procedure in cases in which there is no question of infection, or in other words, in elective operations in clean cases.

Anyone reading Sellheim's paper[1] without having had any actual experience with the usual type of cæsarean operation would certainly conclude that it was an absolutely unjustifiable procedure. After stating that the Poro cæsarean is less dangerous in truly infected cases than the extraperitoneal cæsarean, he enumerates the following appalling list of dangers associated with the former procedure: intestinal wounding, greater loss of blood, loss of procreative power, longer rest in bed after operation, later return

[1] Zentralbl. f. Gynäk.

to work, abdominal hernia, bad cosmetic result, and finally, disturbance of the respiratory function of the child by the forceful forward displacement of the uterus outside the body of the mother, with resultant infection of the child from inspiration or swallowing of infected liquor amnii. Moreover, he believes that there is as much danger of wounding the bladder in the classical operation as in the extraperitoneal, and in addition that there is great danger of infection of the peritoneum, either by soiling at the time of operation or by later leakage from the uterine wound. In addition the uterine cavity may be infected, there may be intestinal or omental adhesions, fixation of the uterus to the anterior abdominal wall, weakening or rupture of the scar in later pregnancy, and abdominal hernia. He says: "Extraperitoneal uterine section is identical with vaginal cæsarean section, all technical difficulties having been removed."

If ever there was an instance of special pleading we have one here. It would have been a perfectly justifiable arraignment of the classical operation if it had been published before the days of the aseptic era, but, in the light of our knowledge of the results which have been obtained in the classical operation in modern times, it is hard to understand how a man could become so blindly enthusiastic about any method, even one bearing his own name, that he could disregard the cold facts as attested by operators throughout the world regarding the Sanger operation in clean cases. Admitting as we have that the extraperitoneal operation is of advantage in a limited group of cases, and further admitting for the sake of argument that in perfectly clean cases, in the hands of men having had experience with this type of operation, the statistics for both mother and baby may, in the future, be as good as those now available in the Sanger operation, we are nevertheless forced to conclude that the burden of proof still rests upon the new operation.

Difficulties in orientation of the bladder, injuries of the bladder, extension of the uterine incision, and increased difficulties in the delivery of the child are factors to be reckoned with in the two methods for the performance of the operation recommended in this paper. If some of the other methods, as Latzko's, be undertaken, we have the added dangers of wounding of the great vessels and the uterers, while the peritoneum is injured in a non-negligible percentage of the reported cases by all true extraperitoneal methods. While it is of course perfectly justifiable to assume risks in order to avoid imminent dangers in obstetric, as in other surgical procedures, it is certainly somewhat illogical to advise the giving up of a method such as the Sanger operation, with its well-established technique, in favor of a procedure which, to say the least, smacks of a return to the pre-antiseptic days. Particularly is this true if we remember that in a series of three hundred cases Leopold was able to report a mortality of 1.2 per cent, that Pestalozza in a series of 111 cases had a mortality of 2.08 per cent (omitting four cases which were infected previous to operation), and that Richter of Dresden in a series of 107 cases was able to report a mortality of 0.9 per cent for both the mothers and the children. This last report of Richter is important from two standpoints; the first, already mentioned, is the actual mortality, while the second appears upon analysis of his series of cases, by which it is found that five of these cases had prolapse of the cord and that fifteen were second operations (of these there were three upon whom the operation of Frank had originally been performed), among whom he found that there were four presenting intestinal and omental adhesions and that in seven instances there were adhesions, mostly slight, between the parietal peritoneum and the uterus. Can any practical man, approaching the subject without creative bias, convince himself that it is justifiable to advocate a procedure which is admittedly, even by its advocates, a much more difficult performance, as a substitute for an operation whose only dangers are those inseparable from all intra-abdominal surgery? In the vast number of cæsarean sections by the usual methods, there are some twenty-six cases on record in which the uterine scar ruptured in subsequent pregnancy.[1] In the operations upon the lower uterine segment there are none as yet, though among the few cases in whom pregnancy has been observed after the suprasymphyseal operation there have been one or two cases in which at the time of the second operation the original scar was so greatly stretched that rupture seemed imminent; but it is admittedly no great danger in either type of operation, on the grounds of the universal experience with the Sanger operation, even with imperfect suture methods, while the knowledge we have lately gained as to the subsequent behavior of the scar after the vaginal cæsarean section will at least allow us to assume the like for the extraperitoneal method. In so far as adhesions are concerned, it is proven that intestinal and omental adhesions of a degree sufficient to interfere markedly with the health or life of the patient are at least rare

[1] Dahlmann, Inaugural Dissertation, Jena,

after the usual cæsarean delivery, but admitting that they do occur rarely and realizing that there can be no intraperitoneal adhesions in a clean case which has been operated upon by the extraperitoneal method, we must not fail to remember that there may be just as troublesome results of an adhesive nature after the operation now under investigation; for it is to be remembered that while there are many points of similarity between the suprasymphyseal and the vaginal cæsarean there are also points of difference, in that in the former there is an abdominal wound which greatly increases the risk of adhesive trouble in the region of the uterus. Whether this last mentioned difficulty is real or imaginary can only be proven by the future.

A very important question which must now be considered is the effect of the extraperitoneal suprasymphyseal section upon subsequent pregnancy, and secondarily whether repeated operation in the lower uterine segment is likely to be more difficult than in the first instance.

It is, of course, too soon to be able to gather a large and convincing mass of statistics bearing upon these points, but still there is enough to enable one to at least form a tentative judgment. Hartmann,[1] reporting from Frank's clinic, is able to publish a most interesting paper upon this subject in which he states that "as early as September, 1908, he had seen one woman twice spontaneously delivered following the extraperitoneal cæsarean by Frank's method." In the same paper he reports upon four cases who had undergone the operation twice and upon two cases who had three times been delivered in this manner. In none of these women were there any difficulties observed during pregnancy and in no case was there a retrodisplacement of the uterus. The first case at the second operation showed a small hernia. She had been drained at the original operation. There were no peritoneal adhesions and no difficulty was experienced in separating the peritoneal folds. There was no uterine scar to be seen and the child was delivered spontaneously through a transverse uterine incision. The second case, after six hours of labor, showed a thinning of the uterine scar suggesting the possibility of rupture, but the operation was completed without difficulty. In the third case there was not only no difficulty but there was no need to do more than push up the peritoneum before making the uterine incision, as the bladder was uncovered. In this case also there was entire absence of any uterine scar. In the fourth case there was some trouble in orientation of the bladder and also in the separation of the peritoneum. This patient developed chills and a urinary fistula subsequent to operation. All these cases had been drained through the abdominal wound and the second operations were all transperitoneal. In the fifth case, one of the two upon whom the operation had been done twice previously, he found a hernia and also some muscle adhesions, and in the sixth case, at the third operation, while there was no hernia there was some scar tissue in the uterine wall at the site of the old incision. Sellheim has reported five cases previously operated by his technique, in three of whom labor progressed to a spontaneuous termination, while in the remaining two he again delivered by his method. In both the operative procedure was more difficult, and the author puts especial emphasis upon this point in several of his articles. In one of the patients there was a widening of the uterine scar, but this was not the case in the other. Mayer reports two cases upon whom an attempted second extraperitoneal operation failed in each instance because of the adhesions between the uterus and the parietal peritoneum. He was compelled finally to resort to the fundal incision to accomplish delivery. In neither of these women was there any stretching of the uterine scar.

Traugott, however, has reported one case in which the scar was about to rupture. Litschkuss (1912) mentions two cases of labor following a previous delivery by Latzko's method, in one of whom birth was spontaneous, while in the other delivery was by the classical operation.

It is thus to be concluded that, in so far as we may judge by the statistics at present available, there is at least no marked tendency to rupture of the old scar or other difficulties in future pregnancy, that labor is not likely to be influenced by adhesions, but that if a second extraperitoneal operation be undertaken it may be found to be impossible and in any event will probably be found more difficult in a large proportion of cases.

In the preparation of this study it seemed to me to be fitting to collect the representative American opinions upon the subject, and I therefore sent out a list of questions to a considerable number of well-known operators in this country. I asked that the questions be answered theoretically if the recipient had no practical experience to offer.

Among the replies which I received there were but three who had had any practical experience, namely, Dr. Dickinson, whose case of elytrotomy has been earlier mentioned, Dr. Howard A. Kelly,

[1] Gynäk. Rundschau, 1909.

and Dr. B. C. Hirst. Dr. Kelly has performed the operation once in a clean case before labor with a good result for both mother and child. He thinks favorably of the procedure and indeed is of the opinion that it may in the future become the method of election in all cases. Dr. Hirst is enthusiastic with regard to the future of the operation. He has used various techniques, namely, those of Bumm, Sellheim, and the Veit-Fromme, the latter with modification, and upon the experience gained by a series of some seven cases he would at the present time use the method except in the severely infected case, to the exclusion of the classical procedure.

With the exception of these two operators the consensus of opinion is absolutely opposed to its performance in clean cases, and in but a small proportion of replies was it admitted that it had any field of usefulness. Some half a dozen men advocated its use in septic cases, there apparently being no very definite understanding as to the true field. But two men essentially agree with me in the opinion expressed in the body of this paper when considering the indications. The general expression of opinion may be epitomized as follows: The difficulties and dangers of the operation are so much greater than those met with in the classical procedure that any possible advantage possessed by the method is nullified. Believing as I do that the operation has a true field though a limited one, and also believing that the future may show that the indications for the operation may be broadened by the advance in our diagnostic ability regarding not only the determination of the variety of microorganism present but its import from the standpoint of virulence, it seems fitting, in view of the attitude of the vast majority of the leaders of the profession in this country, to say something here with regard to the technical difficulties of the extraperitoneal cæsarean section. That it is a more difficult procedure than the ordinary intraperitoneal cæsarean is admitted, but the mere difficulty of an operation, if it has advantages not possessed by any other, cannot be admitted as an argument against it, and in this instance the difficulties have been very much magnified. This I believe is due to the fact that in this country we have been led astray by the insistence of many of the foreign operators upon the extraperitoneal element of the operation, and have not realized that any case suitable for section can as well be done by the transperitoneal as by the extraperitoneal method. That this is true is evident from the fact that in both forms of procedure the uterus is left *in situ*, and if its substance be virulently infected trouble surely will result in the one as in the other; while if there be a virulent infection of the liquor amnii the damage will be greater in the true extraperitoneal than in the transperitoneal since in the former large areas of connective tissue are opened up, while in the latter these same areas are undisturbed. If now, realizing these facts which have been gradually evolved by experience, we consider the difficulties of an operative procedure such as Sellheim's second method or the Veit-Fromme technique, I believe that it will be found that the difficulties are more imaginary than real to the operator experienced in abdominal work. I cannot agree with Sellheim that the operation is identical with the vaginal cæsarean, "all technical difficulties having been removed," or with Frank or Spaeth, who consider it a suitable operation for the general practitioner. I do believe that it is more difficult than is the vaginal cæsarean upon a multiparous woman, and that it is more difficult than the ordinary abdominal cæsarean, and I do not believe that the unskilled man has any business with any of these operations unless under exceptional circumstances of isolation. But for the skilled obstetric surgeon the necessary variations in technique will be easily mastered. I believe that this operation will be performed in this country, for the proper indications, by a rapidly increasing number of surgeons, with gratifying results, and I furthermore believe that it will in all likelihood be performed by the following technique, which is not only relatively, but actually, simple and easy of execution. With the patient in the moderate Trendelenburg position a median longitudinal incision is made from the symphysis to within a couple of inches of the umbilicus. The fascia and the muscles are then separated in the same direction and to the same extent, thus avoiding the use of the Pfannenstiel incision, a distinct advantage in suspect cases, since there can be but little doubt that this incision is more prone to suppuration than the ordinary longitudinal one, and if infected will give a much more serious condition. The parietal peritoneum is then opened by an incision about two inches in length and the upper limit of the mobile portion of the visceral peritoneum is ascertained. This uterine peritoneal layer is then incised from the top of the bladder to the point where it becomes firmly attached to the anterior wall of the uterus. This last mentioned layer of peritoneum is then separated laterally on each side of the median line for a couple of inches throughout the length of the incision. The conditions at this point of the

procedure are as follows: the abdominal wall, exclusive of the parietal layer of the peritoneum, has been incised from the symphysis pubis to a point several inches above, thus giving ample room in which to work; the parietal peritoneum has been incised from the lowest point possible, namely, the top of the bladder, to a point which corresponds to the situation of the lowest area of firmly attached uterine peritoneum; while the uterine peritoneum has been incised to an identical degree. These two peritoneal leaflets are now united by a continuous buttonhole stitch of catgut. An oval opening is thus produced, the upper extremity being formed by the attachment of the free parietal with the agglutinated portion of the uterine peritoneum, while the lower extremity is formed by the union of the anterior and posterior folds of the vesical plica at the upper edge of the bladder. A longitudinal incision in the uterus of about 12 cm. gives ample space for the application of the forceps, which instrument serves the purpose better than the special hooked lever invented by Sellheim. The amount of bleeding is usually not as great as during an intra-abdominal cæsarean, and moreover, in one instance in which it was profuse during the delivery of the child, it ceased at once as soon as the birth was completed, due to the occlusion of the blood-vessels by contraction of the lower segment. The uterine wound is decidedly reduced in size after the delivery, and is closed by a double layer of catgut as in intestinal suture. A continuous catgut stitch closes the stitched folds of the peritoneum and the remainder of the operation is completed in the usual manner. In a suspect case, of course, the necessity for drainage may alter this method of closure. The technique just described is the Veit-Fromme, with the modification that the folds of peritoneum are sewn after their primary incision instead of using clamps to make the closure. One of the great advantages of the method is that the bladder is not displaced, while the use of the median incision through all the layers obviates the dangers of infection of the large areas of tissue exposed by the Pfannenstiel abdominal incision and by the lateral extraperitoneal attack.

I have been able to collect eight cases done by American operators. Seven of these were performed by Dr. Hirst and one by Dr. Kelly. There may be many other cases, and it is to be understood that no claim is made that this is a complete list, but I have been unable to find any other reports either in publications or by personal letter. I will therefore insert a short synopsis of the case histories.

Case 1 (Hirst). This patient was a primipara and had been in labor for twenty-four hours. Her membranes were ruptured when first seen. Examination showed a c. v. of 8 cm. and the head of the child was large. Forceps were applied twice before sending her to hospital. In the first attempt the application was made with due regard to asepsis, but, failing to engage the head, the family physician attempted to deliver by the use of his forceps, which he applied without any sterilization. In this case the Bumm technique was employed. The peritoneum was inadvertently opened but immediately closed, and the mother and child made a good recovery.

Case 2 (Hirst). Had a history of one miscarriage and one child delivered by high forceps at the seventh month. A rhachitic dwarf with a c. v. of 9.25 cm. Several examinations had been made upon her outside of the hospital by the students. Temperature on admission was 99° and her pulse was 112. Had been in labor for 36 hours without engagement and was delivered by Sellheim's first method. Mother and child discharged in good condition.

Case 3 (Hirst). Primipara with a true conjugate of 8.75 cm. In labor for 36 hours before admission; no engagement; was delivered by Sellheim's second method. Had a slight febrile reaction for a few days. Mother and child discharged in good condition.

Case 4 (Hirst). Multipara. All three of her previous labors had been very difficult. Conjugata vera 8 cm. She was a house case and had been in labor for 16 hours, without engagement, at the time of the first pelvic examination. An examination made at this time resulted in rupture of the membranes and prolapse of the cord. She had hard and regular pains. Delivered by Sellheim's second method. Had a badly infected wound giving rise to fever from the second to the ninth day. Both patients discharged in good condition.

Case 5 (Hirst). Multipara. Conjugata vera 8.25 cm. Was a house case and at time of operation the membranes were still intact and the pains had not been severe. Had been in labor about 24 hours. Was delivered by Sellheim's second method and had a moderate infection of the wound with consequent temperature from the second to the ninth day. Both mother and child recovered.

Case 6 (Hirst). Primipara. Had been in labor four or five days. The membranes were not ruptured and there was no engagement. Had a justo minor pelvis of the fœtal type, with marked outlet contraction in both the transverse and posterior sagittal diameters. Was operated by the Sellheim second method and the bladder was injured, but the rent was immediately closed and no trouble resulted. Badly infected wound. Both patients discharged in good condition.

Case 7 (Hirst). Multipara. Conjugata vera 7.5 cm. Head large and high, overlapping the symphysis markedly. Previous history of difficult labors. Was not in labor. In this instance the Veit-Fromme technique was employed with the greatest satisfaction. The lower segment was markedly dilated, due to the high position of the head, and there was ample room to work. Afebrile convalescence with primary wound healing. Child discharged in good condition.

Case 8 (Kelly). Had been delivered of five still-born children. Conjugata vera 6.25 cm. Operation performed before labor had started. Pfannenstiel incision; recti separated but not detached, and only the right half of the bladder was separated. The peritoneum was not opened, as there was ample exposure. Longitudinal incision into lower segment, membranes ruptured and child delivered without forceps. Little bleeding after rupture of membranes and but moderate hæmorrhage on incising the lower segment. Both patients discharged in good condition.

CONCLUSIONS

The extraperitoneal suprasymphyseal cæsarean section is an addition to the obstetric armamentarium.

Its chief indication is the "neglected case" or one that is mildly infected. Since the prognostic differentiation between the various degrees of septic infection is a matter of uncertainty, the operation, in the words of Sellheim, is "of the nature of an experiment in many cases."

The operation is absolutely contra-indicated in the seriously infected case.

The performance of the operation in clean cases is attended with good results in experienced hands; convalescence is more smooth and less hazardous than after the classical procedure.

It is a more difficult operation than the classical, not only in gaining entrance to the uterine cavity, but also in the delivery of the child; the closure of the uterine wound is less difficult than in the Sanger method.

There is less loss of blood than is usual in the classical procedure.

The transperitoneal technique is to be preferred to the true extraperitoneal because of ease of performance, less interference with the bladder, and the avoidance of contamination of the retroperitoneal and preperitoneal tissue areas.

The preferable technique is the modified Veit-Fromme.

Subsequent pregnancy and labor are usually not influenced adversely; repetition of the procedure may be more difficult and in some cases it may be impossible.

The appended bibliography is by no means complete, but simply comprises a few of the more important papers on the subject which have appeared since 1907. It is interesting to note that in the year 1907 there were but four articles noted in the *Jahresbericht für Geburtshilfe und Gynäkologie*. Of these there were two by Veit and two by Frank.

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SOME CAUSES OF FAILURE IN BACTERIN THERAPY[1]

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THERE are many interrelated factors in the production of immunity, and numerous minute details requiring the closest attention, many of which, although apparently insignificant, are really the prime causes of failure. I have attempted to briefly outline and consider the commoner ones and those that experience has shown me are more frequently the cause of trouble with the average practitioner.

We may classify the causes of failure under the following general heads: incorrect bacteriologic diagnosis; improper bacterin, either stock or autogenous; improper dosage, either size of dose or interval between doses; and lack of proper concomitant treatment.

FAILURES DUE TO INCORRECT BACTERIOLOGIC DIAGNOSIS

In some cases it is possible to make a diagnosis clinically, but as the action of bacterins is specific, it necessarily follows that an accurate bacteriologic diagnosis is absolutely essential. With the possible exception of cases of furunculosis, an attempt at a correct bacteriologic diagnosis should always be made. This may, however, be impossible, as for instance where we have a deep-seated abscess or where for other reasons it is not feasible to obtain the infectious material. Further, the material obtained may not show or contain the causative organisms or they may not be recognized, especially where there are several other organisms present. Through lack of bacteriologic knowledge and skill, the causative agent may not be correctly determined. Further, especially in chronic cases of the respiratory tract, the infection changes from time to time, and it is necessary to make frequent examinations and change the bacterin as the infection changes. Any of the above may result in an improper bacterin being given, and necessarily failure must result. This may all be summed up under the head of improper bacterin due to incorrect or impossible bacteriologic diagnosis.

THE BACTERIN

1. *Stock bacterins.* The stock bacterin used may not contain the proper strain. This the manufacturers of stock bacterins have attempted to obviate by producing what are known as "polyvalent bacterins," i.e., a bacterin containing a number of strains of the same organism; but this is in reality only a makeshift, because if ten strains are used and only one of them is the proper strain, the dose given is only one-tenth of that particular strain, the other nine-tenths of the dose being composed of other strains and probably having no effect. While this may seem theoretical yet it is a practical fact. Further, the stock bacterin may not contain all of the bacteria causing the infection — may not be the proper mixture. It is advisable, therefore, in using stock bacterins to prepare your own mixtures. So far as is known, there is no definite disadvantage or objection, from a clinical standpoint, to using a bacterin containing additional varieties of bacteria, except that the patient's cells are called upon to produce antibodies to these additional bacteria, which are without any definite curative value so far as the particular infection from which the patient is suffering is concerned. It may, therefore, be considered as merely an additional tax upon the patient's cells without any direct, immediate benefit.

Failure may also result because a stock bacterin is not properly prepared. It may have been heated too high, so that its immunizing properties have been destroyed, or the bacteria originally used in the preparation of the stock bacterin may not possess immunizing powers. This we find is true in some cases in which old, avirulent stock cultures are used. The stock bacterin may be old, i.e., it may be overkept, as a result of which autolysis has taken place, and in certain infections this bacterin would be of no value, because recovery or immunity depends to a great degree upon the formation of lysins by the cells. If the bacterin has undergone autolysis, this response is not called forth. In certain other cases the bacterin may be underkept, or as we might term it, "green." Autolysis not having taken place, a sufficient amount of endotoxins have not been liberated, so that the formation of anti-endotoxins, the principal immune bodies in some infections, are not as speedily called forth as from the injection of an older bacterin of this same variety.

Again, chemical changes may take place in the bacterin, owing to the dissolving of alkali from the glass, especially if it has been kept under improper conditions, at room temperature. On

[1]Read before the Chicago Gynecological Society, June 27, 1913. (See discussion, p. 262.)

this account it is advisable to use Jena glass, and for this reason bacterins in ampules of Jena glass, or of the new Fiolax glass, which gives up even less alkali than the Jena glass, are to be preferred to bacterins in syringes, which are usually not made of Jena glass.

2. *Autogenous bacterins.* These may not be prepared from the proper bacteria, or the predominating organism may be one that does not grow very readily, and is, therefore, overlooked; or, the bacteria isolated from the infectious material may not possess immunizing powers, and further, the bacterin may not be prepared properly. It may be overheated, etc.

Dose. The dosage may be too small. We therefore do not get a clinical reaction, the negative phase and the corresponding positive phase are very brief, and the immunity does not rise very high. Such a condition does not produce the best results and may actually result in failure, owing to the fact that the stimulation applied is not great enough. Or, the dose may be too large, resulting in the patient's immunizing forces being practically exhausted, with the result that additional large doses cannot call forth a further response. This is to be obviated by careful clinical observation of the patient and the giving of only such a dose as will cause a very slight clinical reaction, not increasing the dose as long as it continues to produce this slight clinical reaction. The frequency of dose varies, of course, depending upon whether the case is acute or chronic, whether a large or small dose be given; failures frequently result from the improper spacing of the dose. Especially large doses should not be given at short intervals. The attempt to produce a summation of positive opsonic phases is the ideal one; and the production of a summation of negative opsonic phases is to be avoided, as it usually results in failure.

Lack of proper concomitant treatment. Lack of proper concomitant treatment has, in my experience, been the cause of the greatest number of failures, at least so far as it has occurred in those cases in which a proper bacterin was used. We may divide this concomitant treatment into general and local. General treatment consists in the proper control of the eliminative organs — renal and intestinal. Constipation, intestinal indigestion, and fermentation produce toxic poisons that have an antiopsonic effect, and their formation and absorption must be prevented. Further, the toxins produced by the infectious agent are destroyed by the spleen and the liver, and are eliminated by the kidneys, skin, and intestines. The intestines are also known to eliminate bacteria, therefore we must keep all of these organs in the best possible working order. One can readily understand that the cells of the body and the organs of elimination may be overtaxed in combating the absorption of fæcal toxic products, or at least so occupied that there is no reserve force capable of being stimulated by the injection of bacterins. Under general treatment also comes the treatment of general diseases other than the infection. We are always to search for certain constitutional diseases in every infection. An example that is familiar to all of us is the tendency to furunculosis in patients with diabetes. The mere injection of bacterins in these cases usually results in failure, and it is only by careful attention to and treatment of the diabetic condition that results may be hoped for. Further, in certain diseases, such as leukæmia and leucocythæmia, the leucocytes possess very little phagocytic power, and it is possible that although you may produce a high opsonic index in patients with these diseases, recovery will not occur, owing to the inefficiency of the leucocytes.

Local treatment aims at removing foreign bodies and sequestra harboring the infectious agent, otherwise continued reinfection occurs. Healing will not take place as long as there is an infected foreign body in the tissues, so that surgical measures wherever indicated require prompt attention. Then, we may have a high opsonic index content of the general serum, but the local circulation may be sluggish, or owing to local high pressure conditions, the opsonins may not have access to the local lesion. This is to be remedied by free drainage, or such measures as tend to produce a local hyperæmia — the use of hot fomentations, Bier's hyperæmia, Finsen light, manipulation, massage, etc. These, of course, will all tend to produce auto-inoculation, and in certain cases are to be avoided. Especially is this true in tuberculosis. The patient should be placed in bed at absolute rest, as exercise results in auto-inoculation, which, if continued, will have practically the same effect as a too frequently administered dose of bacterin.

The local use of antiseptics must be governed by the following rules: a sufficiently concentrated germicide must be used to destroy all of the bacteria, leaving a sterile site, otherwise the destruction of tissue will merely augment the further action of any bacteria that have not been destroyed; or, a mild antiseptic may be used, sufficient to inhibit the multiplication of the bacteria but not sufficient to destroy the tissues. Preferably, antiseptic solutions are to be sup-

planted by the use of Wright's 5 per cent sodium chloride, sodium citrate solution. This prevents coagulation and augments osmosis, thereby promoting the flow of lymph with its increased opsonin content. The lymphatic circulation may also be enhanced by the administration of citric acid, 30 to 60 grains three times a day, as advised by Wright.

There still remains a possible cause of failure in the lack of a sufficient number of active leucocytes. Exactly how this may be remedied is not yet definitely known. We can increase the number of leucocytes by the administration of nuclein, sodium cinnimate, pilocarpine, or salicylic acid, but we have no knowledge or direct evidence to prove that the phagocytic power of these leucocytes is normal. Personally I have used nuclein with apparent clinical success. This seems to me to be the most rational of the drugs mentioned, as it is a constituent of the leucocytes themselves.

Failure may possibly also result from an improper selection of the site of injection. Our present knowledge would indicate that subcutaneous or intramuscular injections produce the best results. Wright and Allen do not believe that intravenous injections are of value. They reason that the blood is not actively engaged in the production of antibodies, therefore the circulating streptococci in septicæmias do not result in the production of immunity. That this is not a general rule, however, and will have to be definitely worked out for each organism, has been proven recently by the fact that in contagious abortion of cattle the best results are obtained by the intravenous injection of the bacterin. Whether this holds true for any other organisms, we do not yet know.

There are some cases in which failure results for reasons as yet unknown. It is very evident that all infections, or all persons, irrespective of their infection, are not amenable to bacterin treatment. Some are incapable of responding. Bacterins will not produce a cure in all cases, and the tendency of the times, owing to the apparently wonderful successes, is toward overenthusiasm. We are repeating with bacterins the history of tuberculin — using them for everything, irrespective of the indications, and usually with a free hand, often without any definite reason, and in any disease, whether it be a proven infection or not. Cases are being reported of wonderful success; very frequently the number of cases is not mentioned, and the average clinician, without opsonic or bacteriologic control, is using any bacterin, in every sort of disease, and reporting wonderful results in one or two cases. He becomes overenthused, and passes this enthusiasm on to others, but many times they are doomed to failure. This is a very unfortunate condition, for bacterins are of value, if thoroughly understood and rightly used, and do produce results in many cases that are nothing short of wonderful. I sincerely hope that before long they will be sufficiently understood, their indications and limitations known and recognized. They will then obtain and maintain their proper place in therapeutics.

NEW APPLIANCE FOR THE INTERNAL FIXATION OF FRACTURES OF THE FEMORAL NECK

By M. E. PRESTON, B. A., M. D., Denver, Colorado

FRACTURES of the neck of the femur, for years, have been among the most difficult we have had to treat. Operative intervention in these cases has not been satisfactory because of the difficulty of holding the fragments in position even after they have been exposed. The reason for this difficulty is most apparent if we study the internal structure of the upper end of the femur. The illustrations of sections of the upper end of the femur (Figs. 1 and 2) give a good idea of the relative disposition of the compact and cancellous tissue in the femoral neck.

When, during operation, reduction is temporarily accomplished by traction and manipulation, and an ordinary screw is driven through the trochanter and neck, the fixation thus accomplished is practically worthless. As soon as upward traction is exerted on the shaft by the heavy muscles of the thigh and hip, the cancellous tissue on which the screw takes its hold, crushes, and the usual deformity follows (Fig. 4.) A röntgenographic study of the cases operated on in this manner will show the inefficiency of securing the fragments by this method. It is granted that

good results have followed this operation, but I am satisfied that in most instances they have been due more to the action of a properly applied Buck's extension during the after-treatment than to the securing of the fragments by means of the ordinary screw or nail. The essential reason for this failure in fixation is due to the fact that the ordinary screw or nail used for this purpose secures its hold only in cancellous tissue.

To obviate this difficulty I have devised a screw-plate which secures its hold on the compact tissue below the great trochanter and will maintain the normal angle of the neck in spite of the action of the thigh and hip muscles. It is a combination of a screw and a plate in one piece. The screw portion is of the ordinary wood type and is made in varying lengths while the plate part is fashioned after the Lane plate. The screw portion joins the plate at about 110 degrees, which is less than the angle at which the femoral neck joins the shaft. This difference is due to the fact that the surface of the shaft just below the trochanter is not parallel with the axis of the femoral shaft, and since the plate secures its hold on this portion of the bone, the angle has been adapted accordingly.

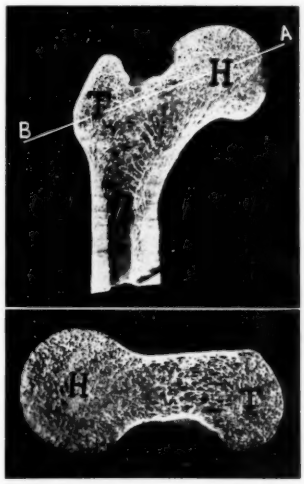

Fig. 1. Coronal section of upper end of femur showing the relative disposition of compact and cancellous tissue.

Fig. 2. Section of femur corresponding with the line A–B in the preceding figure.

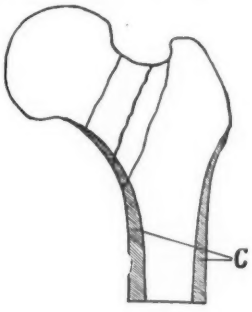

Fig. 3.

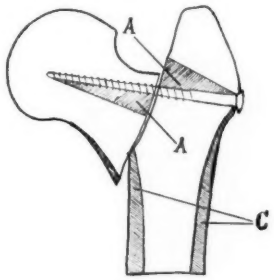

Fig. 4.

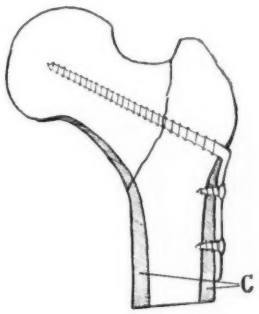

Fig. 5.

Fig. 3. Diagram of femur showing the common sites of fracture of the femoral neck.

Fig. 4. Diagram showing the manner in which the cancellous tissue crushes when the neck is subjected to the displacing action of the thigh and hip muscles, following operation with a simple screw or nail.

Fig. 5. Diagram showing the author's screw-plate in position. Angulation of the screw portion is prevented by the plate portion which secures a firm hold on the only available compact tissue in this region.

The length of the screw portion varies with the size of the bone and the position of the fracture. When the break is near the inner end of the neck, the screw should extend well into the head of the bone. When the fracture is situated at the base of the neck a shorter screw will accomplish the desired fixation. A number of sizes should be at hand so the surgeon may select the proper length.

The angle at which the plate joins the screw is such that the plate clears everything as the appliance is being screwed into position. Before the screw is entirely driven home the plate should be bent with pliers so it will fit the surface of the femur with which it will ultimately come in contact.

I have not as yet used the appliance in a sufficient number of cases to give a report which would be particularly valuable or conclusive, yet the mechanical fitness and ease of application of this screw-plate have demonstrated its value, pending a further report.

TRANSACTIONS OF SOCIETIES

CHICAGO GYNECOLOGICAL SOCIETY

A REGULAR MEETING WAS HELD JUNE 27, 1913, WITH DR. FRANK W. LYNCH IN THE CHAIR

CARCINOMA OF THE APPENDIX

DR. EMIL RIES: I have some rare specimens that may interest the members. The first one is a case of carcinoma of the appendix. It is the first one I have had, and I want to say I got it entirely accidentally. I opened the abdomen of this patient, a woman 40 years of age, on account of chronic salpingitis, with adhesions, and we supposed that her intestinal difficulty, constipation and so on, were due mainly to the adhesions. When we opened the abdomen, freed the adhesions and removed the pus-tubes, which she had, I looked at the appendix and found a perfectly solid structure. This appendix is cut open in several places and you can see on the cut-surface how the muscular coat of the appendix is contracted, and the center of the organ is solid and is protruding through the sheath of muscular tissue. This is entirely different from what we are accustomed to see in cases of obliteration of the appendix, and the feel of the appendix should immediately tell us this is not an ordinary obliterated appendix. The ordinary obliterated appendix, when it is cut open, shows a tendency to retract in the center because the slight amount of cicatricial connective tissue that forms there is more liable to draw back than to push out. Here the center is pushed forward. It evidently is under more or less pressure. It was clear this woman had something different from the ordinary obliteration of the appendix, and the microscope confirms the diagnosis of a malignant neoplasm.

It is not absolutely certain whether it is an adenocarcinoma or an endothelioma. There are few traces of lymphoid tissue. The rest of the appendix, except the muscular coat, is taken up by a net-work and a mesh-work of epithelial, or epithelioid, cells which are arranged in files of one or two; then they run together in large masses of cells, and these larger masses contain vacant spaces or vacuoles which remind one of the formation of endothelioma. I have placed under the microscope one part of the appendix which shows this mass of endothelial neoplasm very distinctly.

I have made dozens of sections and find in various parts of the appendix that the endothelial neoplasm apparently does not reach outside of the muscular coat. It is found within the muscular coat completely. It is not the nature of carcinoma to respect the muscular coat in any way. It is the nature of carcinoma to break through it. I found a number of sections in which the carcinoma had broken through the muscular coat and was in the serosa of the appendix. I have sections which show the carcinoma in the serosa of the appendix.

Carcinoma of the appendix occurs in two forms: one the solid form, and the other the form in which a large amount of mucus is produced. You doubtless remember the case of Dr. Watkins, which was in the shape of a cyst. That cyst proved to be a carcinoma of the appendix, with the production of so much mucus that it had distended the appendix. These are the two forms, the present case being the solid form in which you can see solid masses of neoplasm very distinctly.

Here is the carcinoma in the serosa. As far as one can tell in the living, I have investigated the gall-bladder and stomach and intestines as far as I could, and there is no tumor anywhere else. I removed the appendix without leaving any stump. I excised the appendix at the cæcum, made a fair sized hole in the cæcum, and sewed up the hole and extirpated the whole structure. The patient has had no return or trouble of any kind.

Cases of carcinoma of the appendix are interesting in this particular, that the tumor is rather benign. There are a considerable number of cases on record in which surgeons have gone into the abdomen and accidentally found carcinoma of the appendix. This woman had no symptoms of appendicitis or of any tumor. There are no metastatic glands anywhere. She is in perfect health so far. The operation was done five months ago.

STRUMA OF THE OVARY

DR. EMIL RIES: I have here another specimen of a case of struma of the ovary. I removed from a woman an ovarian tumor of large size, which was filled with colloid material which contained apparently one cyst. In the wall of that cyst was one rather hard nodule the size, I should say, of a 50-cent piece, and it was about one-half inch thick. Sections through the thin part of the cyst showed nothing but an ordinary thick wall, and I found no epithelium on it, as we do in so many cases when the cyst has been very much stretched. But in the solid part I found a peculiar condition. I found the solid part to consist, first of all, of a material which in the hardening process coagulated to the consistency of almost celluloidin, and it appeared like the colloid of the thyroid gland. When we made the sections we found it was typical thyroid gland, the normal epithelium of the thyroid, but the colloid in the alveoli of the gland is of various kinds. You know that the colloid of the thyroid gland does not always stain the same way in the same gland. Some parts take more of the eosin stain, and some more of the hæmatoxylin stain, and with Van Gieson's stain we get various colors; this was true of this gland. In certain larger cyst-cavities of the more solid

portion of the tumor we found longitudinal slits which are characteristic of cholesterin which has been dissolved out in making the celluloidin preparation. The microscope shows the typical thyroid structure and the thickness of the tumor-wall. The last gland I removed from the neck. How does the thyroid get into the ovary? The simplest explanation is that there is a growth of the thyroid the same as there is a growth of other derivatives of cells which by an error of development get into the ovary. In all these cases we had to examine carefully to see whether there were any other structures except that one in the ovary, in order to characterize the tumor as embryonal or the derivative of more than the germinal epithelium. I have examined a considerable number of sections of this tumor, and I have found nothing else except thyroid tissue. Of course, when we find thyroid tissue, the next thing to look for is cartilage of the bronchial tree. We found no cartilaginous cells. There is no structure except thyroid tissue. There is nothing of any other organ, so that we have here one of these rare cases. There are twenty on record, all developing in one organ, only in an aberrant way and in an abnormal locality. This is a parallel case to the case of another practitioner, in which a single tooth was found in the ovary. In that dermoid structure there was just one tooth. In the same way just a thyroid gland developed here.

The two specimens, the one from the ovary and the other from the neck, show the thyroid tissue and cholesterin in the same field. This development of cholesterin in the colloid of the thyroid is nothing peculiar to the ovarian stroma. It is found in the stromas of the neck as well.

I have a number of thyroids which I have removed which show this development of cholesterin. In one case in which I found it more than in any other the patient had a thyroid fistula. This fistula led down to the thyroid gland, and had developed after an attack of typhoid fever. The patient had typhoid fever, and contracted an infection of the thyroid from the typhoid. A doctor opened up his typhoid strumitis, and evacuated a large amount of pus, but the abscess-cavity did not close. He had a discharge from the cavity. We took some of the discharge from the thyroid, made a Widal test and found it was positive. This cavity contained bony calcified masses, such as those described in Kocher's paper; and, as all these fistulæ are arranged, this fistula extended downward, and that is one of the reasons why it did not close. The deepest part of the fistula existed when the man was standing up. The thyroid was dissected away from the spinal column and extended to the side of the œsophagus. I injured the jugular vein and the thoracic duct. It was all one mass of adhesions and fibrous tissue. The thoracic duct discharged the customary amount of milk for five days, and then the patient got along all right. In that thyroid there was a vast amount of cholesterin. In some cavities, if you open the thyroid, you can see the cholesterin glisten. If you shake the fluid you can see the light reflected in these cholesterin crystals.

DISCUSSION

DR. JONES: Do you not consider it very important to make sure of the diagnosis as to whether it is endothelioma or adenocarcinoma, as having a bearing particularly on the prognosis? Usually it is better to determine which it is.

DR. RIES: I do not believe that we can say there is very much difference in the malignancy of endothelioma and the malignancy of adenocarcinoma. I think it is six of one and half a dozen of the other. At first, one would say this is a carcinoma, until you have examined the specimen and found vacuoles, perfectly round, without mucous secretion. You are reminded more of an endothelioma than an adenocarcinoma. I think it would be rather fruitless to quarrel over the differential diagnosis. I would rather leave it to a better pathologist than I am. As far as the prognosis is concerned, I do not see that there is much difference.

DR. JONES: Zeit and others hold that endothelioma is not so malignant as carcinoma; that it occurs locally and metastasis occurs late, which is not true of endothelioma, particularly of the lymphatic type.

DR. RIES: A case in which I saw the largest amount of metastasis was one of endothelioma of the uterus which Zeit described in his paper on endothelioma. That case had enormous metastases, in such incredible locations and in such enormous amounts, that it was an exceptional instance. The microscopic slides proved it was an endothelioma of the uterus. There was no ulceration but there was hæmorrhage. An exploratory incision was made into the cervix, in which we found endothelioma. The patient did well. She went on a trip to Europe, and four months after she left the hospital I got a letter from her from Naples stating that she had rheumatism in the knee. The attack of rheumatism spoiled her trip. She returned to America. After her return, in getting out of bed she broke her thigh, leaving a large bony tumor sticking out at the hip. This woman soon showed local recurrence, especially in the bladder, and died in less than two years after operation. She was a mother and the wife of a physician and understood the value of post-mortem examinations, and left word with her son that I was to be notified when she died. I was notified within half an hour after her death. I made a post-mortem examination. This woman had metastases in various organs of the body. She had in the upper end of the vagina one mass of tumor which had broken through into the bladder. The pelvic connective tissue was one tumor-mass. The tumor involved the wall of the rectum. The large so-called bony mass that stuck out at the hip was an enormous carcinoma of the sciatic plexus as large as a fist. I have a section showing endothelioma between the nerve-fibers. The femur was not broken. I cut the femur open. There was no fracture of the femur. She had metastases in the kidney and spleen. The liver weighed 8 pounds. She had metastases in the adrenals, in the lungs, the pericardium, and the retroperitoneal glands. In short, there was

not an organ in the body that did not have some metastasis. We do not see endotheliomas frequently. They are comparatively rare in comparison with carcinomas.

CHORIONIC VILLI

DR. RIES: A few weeks ago I showed sections and a gross specimen of a case which I diagnosed as chorionic villi remaining in the uterine wall eighteen years after the last labor. For those who were not present at the time I may recall a few facts. This woman had a fibroid tumor. I operated on the fibroid and removed it by supravaginal amputation. The operation was not attended with any difficulty whatsoever. When I was through with the operation I proceeded to investigate the specimen and noticed on the cut-surface, where the body of the uterus was cut away from the cervix, a whitish string hanging out of the cut-surface. Following up this whitish string I found it extended into a vein, and found that this vein extended to the length of from two to six inches, so that the upper end of the string was in a vein in the right uterine horn when the cut-surface was that far away from the right uterine horn. In cutting through the uterine wall I found other veins filled with smaller white strings, and on grasping some of these strings with tissue-forceps I found I could pull them out in a sort of bunch, like grapes, which reminded me of chorionic villi. I made microscopic sections which showed the shape of the white strings which filled the vein, and found them to be composed of chorionic villi, round, elliptical, branching, ramifying. This substance consisted of fibrous tissue with blood-vessels, the blood-vessels containing blood-cells the same as the rest of the uterus contained. The epithelium was covered with these villus-like masses. The endothelium lay flat. Each villus was apparently attached to the wall of the vein in some way, and where it was attached to the wall of the vein it perforated the wall of the vein, rupturing the circular muscular coat and penetrating the surrounding tissue. This tissue showed more or less homogeneous fibrous structure going through the vessel-wall into the uterine section. When I showed the sections at a previous meeting some of the gentlemen differed. One could see a double layer of epithelium. Another one could see syncytium. I could not see that. I still have been unable to find any syncytium. I wish I could find syncytium; then there would not be any doubt of these things being chorionic villi.

I did not say anything about what had been found in the literature regarding similar structures in certain cases of chorio-epithelioma. Dr. Robert T. Frank, of New York, wrote me a letter raising the question as to whether it could be proven absolutely that these were chorionic villi, or if it was not possible this was one of those comparatively rare cases of "vein myomas." He himself had a case of chorio-epithelioma in the testicle of a man in whom these white masses had grown into the mass and filled the veins to a greater or less extent. This structure is not distinctly that of chorionic villi the same as in my case, because there was no distinct syncytium covering the apparent villi.

What is to be said in favor of the idea that these things are villi? First, their shape. What may be said against the idea that they are villi? First, the remarkable length of some of these clefts. Who has ever seen a villus that long? Of course, it is possible that this long string which I found in one of the veins was not one villus but a whole villous tree, which became squeezed into the vein, the lateral branches of which were partially obliterated, so that this is the original part of the chorionic tree.

I am rather inclined to think the idea of calling these things "vein myomas" gives us an easier explanation, especially for these very long ones. The chorionic villi which were attached to the wall of the vein showed perforation of the vein circumference, and the circular layer of muscular tissue forming the vein in such cases is against the nature of these things being "vein myomas" because "vein myomas" originate in the muscular layers of the vein. Until we have a definite characteristic of the difference between "vein myomas" and chorionic villi remaining in the vein, the matter must be left in a state of suspension.

As I have said, I have not been able to find syncytium, which one of the doctors believed he saw, and until I recognize the force of the conclusion that such formations may be due to "vein myomas," we must regard the matter as uncertain. However, until the matter is cleared up we must leave the case reported in the literature as one of chorionic villi remaining in the uterine wall eighteen years after the last labor. I have considered it my duty to make a report in which I mentioned Dr. Frank's letter and the importance of this discussion of this case.

DISCUSSION

DR. FRANK A. STAHL: Dr. Ries has brought out the point that he was not able to find syncytium in the specimen that he presented at a previous meeting of the society. Dr. Webster was rather inclined to the opinion that this was one of the points of differentiation, and that this specimen showed the development of chorionic villi characteristic of a certain period. This is a familiar subject, and we all know that in the later months of villous development we have the absence of syncytium, so-called. I presume it is called syncytium from the so-called immediate action of the cell in its environmental tissue. Later on in its development the muciparous limiting stroma disappears more or less and you have practically a granular limiting membrane, which I do not think is a limited membrane, but which is merely muciparous stroma mixed from the absorption of the granules. Its surrounding tissue is broken down by the amœbic action of Langerhans nuclei or cells. In the specimen we still

have these brown granules which are characteristic of syncytium.

I would like to call attention to the fact that in the specimen exhibited that evening there was a limiting structure which was characteristic of syncytium so-called.

As regards the length of development, in the report of a two weeks' specimen of extra-uterine pregnancy I showed a villus which extends from this small chorion down to the inflammatory exudate in the ampulla of the tube. Instead of developing in the nature of a string-condition of the villus, which is characteristic of villi in cases of extra-uterine pregnancy, where you have a club-shaped, juicy, rapidly growing expression of the root, there we had a string-like formation. It was impossible for us to obtrude upon the generosity of the editor, otherwise I might have presented a case of chorionic villus in that small type of gestation which would apparently approach the suggestion Dr. Ries has made here this evening. It was a long drawn out string, but as it was only a two weeks' pregnancy I removed it at the end of 8 or 9 days. As a comparison, it would be favorable to Dr. Ries' string of the same order, and it grew in a perpendicular rather than in a longitudinal direction instead of budding out in all directions.

As regards the venous myoma structure, I think we had better leave the subject just as Dr. Ries mentioned it, but the specimen surely presents a villous development such as we suggested the other evening.

It is gratifying to me, Dr. Ries, that all your work would seem to be confirmatory of the suggestion I made in my work at that time, not thinking it would be possible to have it confirmed by a member of our society. In my reprint I showed you the nuclei, the external layer wandering into all coats of the tube.

THE PERSISTENCE OF GONORRHŒAL ARTHRITIS IN THE PRESENCE OF CHRONIC GONORRHŒAL INFECTION IN THE FEMALE GENITALIA

DR. D. B. PHEMISTER: Both of the cases I wish to report are instances of joint-infection, one gonorrhœal and the other tuberculous. I will present the first case of gonorrhœal infection. The patient was a female, eighteen years of age, who has had trouble with her joints for eleven years. When she was seven years of age, she was taken with rather acute symptoms in the right knee-joint, followed by an effusion into the joint; and this developed into a chronic condition which persisted for about a year. Finally, a diagnosis of tuberculosis of the joints was made and the patient was treated with a plaster of Paris cast for two years. Then from about the ninth to the fourteenth year she had recurrent attacks of effusion into the joints and was on crutches most of the time. From the fourteenth year on, for the last four years her right knee-joint did not bother her very much. It became rather quiescent, but there was considerable stiffness in the joint. Three years ago the left joint became involved, and during the last three years she has had repeated attacks of pain in the joint, followed by a marked serous effusion and pain which kept her off of her feet three or four days at a time. She would improve, and after a few weeks the effusion in the joint would disappear to be followed by a recurrence. An examination of her at the time showed in the right knee, which was involved for eleven years, a moderate amount of thickness about the joint. There was a restriction of motion to about one-half in the joint, but only a small amount of tenderness on manipulation. Examination of the left knee-joint showed a very marked deviation and just mild inflammatory signs in the joint. The first thing I did, after making this physical examination and finding evidences of chronic arthritis in the joints, was to have X-ray pictures made of the joints. From the X-ray plates I was able to see that almost certainly the process was due to gonorrhœa. I want to show you these plates of the left knee, where the process was only of three years' standing. They showed practically no changes in the bones. The plates of the right knee show no local atrophy, but they show an undermining of the cartilage of the epiphysis of the tibia. This is a finding which is quite characteristic of either. It is a condition which, when we find it, is nearly always due to either tuberculosis or to gonorrhœa; and in addition to this undermining of the cartilages on the sides there was an arrested growth of the epiphysis in its anteroposterior diameter. This arrested growth is due to the fact that there has been a chronic arthritis, with restriction of motion, and the limb had not gone through the normal range of motion, and for that reason was not more than one-half as thick in its anteroposterior diameter as it should be.

These lines on the side are practically always due to either tuberculosis or gonorrhœa. If this was due to tuberculosis, there would be extensive destructive changes there. The fact that the left knee was not involved after three years practically rules out tuberculosis, because there would be extensive destructive changes there in that case.

After this physical examination and the X-ray examination, I felt practically sure the trouble was due to gonorrhœa, and I got some more of the history. I found that when the child was six and a half years of age she developed a vulvovaginitis, and within six months afterwards the right knee became involved. I got a history that with every exacerbation in the joints, even before puberty, she would have leucorrhœa. As the general condition quieted down the leg would improve, but sooner or later there would be a recurrence of the trouble. Since puberty she has had more leucorrhœa, with the joint-attacks, and she has also had occasional attacks of pain. Last fall she had two attacks of what we diagnosed as appendicitis. Then I made a pelvic examination. I found a mass to the right of the uterus. As it was nothing that I could feel I left it. Then I called in Dr. Irons, and he made a series of laboratory tests. He made the fixation test, the allergic reactions, and got a positive skin reaction to the gonococcus but a negative one of all the other organisms. He examined the blood, and a Wassermann was negative, but there was a complement-fixation for the gonoccocus. He tapped the knee-joint and got a serous fluid from which he made cultures and recovered the gonococcus from the knee-joint. That verified the diagnosis. The child had had gonorrhœal pelvic infection when six and a half years of age, with involvement of the right knee-joint lasting for eleven years. The involvement of the left knee-joint began eight years afterward and lasted up to the present time.

The interesting features about the case are the persistence of the joint-infection and the inter-

mittent recurrence or intermittency of the process in the joints.

Another interesting feature was the late involvement of the left knee-joint; then the presence of a tumor-mass in the pelvis. In gonorrhœal infections in children, involvement of the tubes leaving a mass is very unusual, and it is a question if the tubular involvement is not a recent condition, either due to a lighting up of the old infection or possibly to a secondary infection, that is, of an acquired gonorrhœa in the last year and a half or two years. The patient gives no history suggestive of this, but it may be in the same way that the joints light up, so may the pelvic process occur, thus causing this tubular involvement of the right side. The persistence of the process in the knee-joints is, in all probability, due to the fact that the pelvic condition has never cleared up. If the pelvic condition had cleared up in all probability the process in the joints would have disappeared long ago. We know that this is the case with metastatic gonorrhœal infections in other locations, and it is the case in the male. A chronic gonorrhœal prostatitis is responsible for the persistence of gonorrhœal arthritis and for the recurrence of the process in the same way in infections of the uveal tract and of many other locations.

I think the ideal treatment, if we could have carried it out, would have been to remove the adnexa; but it is a question if removing the tubes alone would have been sufficient. Probably a panhysterectomy would have been necessary to get rid of the pelvic infection, and then the gonorrhœal arthritis would, in all probability, have cleared up.

The second case I will relate briefly to show the comparative value of the laboratory findings in the two cases.

The second case was a twenty-three year-old Polish woman from whom we got a very imperfect history. She was married at the age of eighteen. She had had no miscarriages and no children. Seven years ago she was taken with arthritis of the right knee-joint and has been bothered with it ever since. In this case the process is much more severe than in the other. She has been on crutches most of the time. Two years ago she developed abdominal and pelvic symptoms, the exact nature of which we did not ascertain. She was operated on at the Cook County Hospital, a laparotomy being done. She came to us complaining of the knee. She was on crutches. She had a stiff and swollen knee, and also pelvic pain. Examination showed a very much thickened knee-joint, with a small amount of effusion, and an X-ray examination of the joint showed that very destructive changes had taken place. It is easy to see the striking difference between the joints in this case and in the other due to gonorrhœa.

The semilunar cartilages are entirely destroyed. The cartilage of the ends of the bones is destroyed and there is considerable destruction of the bone itself. There is marked local atrophy, so that the X-ray findings in this case seem to indicate tuberculosis. The pelvic examination showed the uterus to be retroverted, with a firm tumor on the right side. Dr. Irons made laboratory tests in this case. The skin-reactions were negative. I will say that the von Pirquet was not made, unfortunately, but for the gonococcus and all of the other organisms the skin-reactions were negative. All of the blood-reactions were negative. There was no Wassermann, no complement-fixation of the gonococcus. The fluid from the knee-joint showed nothing culturally.

On the strength of these findings we made a diagnosis of tuberculosis of the knee-joint, and the diagnosis as to the pelvic condition, while it was not positive, was supposedly a tuberculous process.

Dr. Heaney opened the abdomen and found tuberculosis of the tubes, ovaries, and of the endometrium and a healing tuberculous peritonitis. The knee-joint was resected and we found very extensive tuberculosis of the knee-joint in the ends of the bones.

As in the other case, the process started in the pelvis and extended to the joints and persisted in the joints because of the persistence of the process in the pelvis. The process in this case started in the right knee-joint, persisted for years, producing very extensive changes, and then gave rise secondarily to a tuberculous peritonitis of pelvic involvement. It was the reverse of the other condition. While the pelvic condition in the other case gave rise to joint-involvement, here a neglect of the general condition gave rise to pelvic involvement.

I will show you the specimens of tuberculosis of the knee and tuberculosis of the genitalia.

Microscopically there is involvement of the endometrium, complete destruction of the mucosa of the tubes, which is fatal with tuberculous disease, and then the process in the ovary was just a typical tuberculosis. There was no involvement of the muscle of the uterus in this case.

DISCUSSION

DR. ERNEST E. IRONS: An interesting feature in this case was the recurrent character of the

arthritis. It is true that in these cases the arthritis was not recurrent at any definite period of a certain number of days or weeks, but in other ways the case was similar to the one which has been called intermittent hydrops of the knee. There are recurrent attacks of arthritis, swelling, effusion, temporary disability, leaving a relatively intact knee, such as one sometimes notices in an intermittent hydrops in which practically the only difference clinically is that in the intermittent hydrops we have a definite cycle or day.

We had recently another case of true intermittent hydrops with a ten-day cycle in which we were able to study the various reactions of the blood and skin, and in which in one of the recurrences of the hydrops we were able to isolate the gonococcus. I know that there are many cases probably of intermittent hydrops that are not due to the gonococcus; but from a study of three or four cases we have been able to make in the last four or five years, of gonococcal origin, it would seem that a considerable proportion of the intermittent hydrops cases are really of gonococcal origin. In this particular case of intermittent hydrops we were able to make out that there was a very definite cycle of immunity which had a definite relation to the occurrence of arthritis. We know that if we inoculate a case of tuberculosis with tuberculin we get quite a marked reaction of the skin. The same is true to a less degree of certain other infections, including preparations made from the gonococcus. If we had a suitable extract of gonococcus and inoculated it under the skin, we would get, in a case of gonococcal infection, a certain degree of reddening of the skin in much less degree than in the ordinary von Pirquet, but having a definite character. Applying that test to intermittent hydrops, you get a very definite variation in the degree of skin-reaction from day to day. Two or three days before the recurrence of the attack you get practically no reaction, and then a little later, after the attack has occurred and the knee is full of fluid, you get an increased reaction, so that the reaction now is three or four times as great as during the low period. If we take the blood from such a patient every day and test it for the complement-fixation for gonococcus, we get the same period of low activity. A little later, when the joint appears to get better, we get a positive complement-fixation, and these reactions can be shown to follow a definite cycle corresponding to the recurrent cycle in hydrops. These findings possibly explain why we have recurrent hydrops. It is a question of immunity of temporary character which is stimulated by the recurrence of arthritis and subsides promptly. Such a view fits in with the clinical cases of gonoccocal infections. One attack does not protect against another attack. The cycle of immunity is there and is short, and these laboratory findings confirm that view. It is characteristic of gonococcal infection. It is quite different from what we have in typhoid fever, where the immunity develops more slowly.

As far as the diagnostic value of the complement-fixation test is concerned in these two cases, and judging from painful experiences in a large number of other cases which have been proved clinically, we should feel that the complement-fixation test in cases of gonococcal infection is unquestionably of decided value.

So far as the cutaneous test is concerned, it is of great value in tracing the curve of immunity. It probably will never be of great value from the standpoint of diagnosis, because the number of exceptions must be considered.

Dr. Joseph Biehn read a paper (by invitation), entitled "Causes of Failure in Bacterin Therapy." (See p. 258.)

DISCUSSION

Dr. Irons: I would like to emphasize the last paragraph of Dr. Biehn's paper as to the indiscriminate use of the attempt to cover all forms of infections by specific therapy, and to say that such a method is in great danger of bringing this form of therapy into entire disrepute. It is a method that any of us feel has definite possibilities and still greater opportunities for the future, but requires careful study on the part of those who are using bacterins.

A Member: I would like to call attention to the use of nucleinic acid. We tried it in a number of cases and watched the leucocytosis to see if we got any reaction from it; and I would like to ask the author of the paper if there have been any further laboratory contributions showing that nucleinic acid is of value, not from a clinical standpoint, which is sometimes misleading, but from the standpoint of laboratory tests, either by examinations of the number of leucocytes or by their activity.

Dr. Ries: Perhaps this is an opportune time to try to elicit a little discussion among the members and guests as to their experience with a certain new product which is very actively advertised and more or less discussed in the periodicals but which is not really a bacterin, but for which the most marvelous results have been claimed. I am speaking of so-called phylacogen. If there is any grain of truth in the vast amount of advertising literature that is being put out regarding this agent, I should like to have it tested.

It is claimed that the preparation of these phylacogens is not secret. It is claimed that it is positively demonstrable as to what goes into these phylacogens. The theory on which the treatment is built up is entirely fanciful. There is no scientific foundation for it. I cannot understand how anybody with any regard for his scientific conscience can be willing to use such a "gunshot" prescription as these phylacogens are. Nevertheless, we hear of such wonderful successes from the use of phylacogen in certain diseases — and I would mention especially rheumatism and pneumonia — that it would be very interesting, aside from all possible theoretical foundations, to know how much of actual clinical facts have come to the knowledge of the members of this society. I regret I am unable to make up my mind to experiment with this drug, this mysterious substance, but I have been an interested spectator in at least one case of puerperal pyæmia in which a friend of mine gave phylacogen to the patient in the way advised by the firm preparing the substance; and I wish to state that the outcome of that case was in no wise different from the way these cases usually end. That is to say, after the patient had a considerable number of chills, and then phylacogen was administered, and she had some more chills, and phylacogen was then discontinued, and she had some more chills, she ultimately got well the same as we see any other cases get well that have had no treatment at all except alcohol, or hot or cold baths, or no douches, or some douches. In other words, without any treatment, I would like to know if any of the members have had experience with phylacogen. I would like to hear from any member, and particularly from some one who has had a negative experience. We all know the opsonic treatment of puerperal infection has been a sad failure. I do not know of any series of cases treated according to the Wright method that show in any way anything better than we can accomplish by ordinary nursing. I do not know of any series of cases in which any of the modern bacteriotherapeutic methods have given any better results than we usually accomplish to-day with ordinary nursing methods.

This is a subject of the greatest importance in view of the utter impotence which characterizes the eminent gynecologists and obstetricians in the presence of cases of puerperal sepsis, and it would be most desirable to make use of some of the successful methods of these modern investigations. If any one has had success I wish he would speak out loud and tell us. Speak of his failures, so that we may confirm our opinions.

DR. BIEHN (closing): As to the use of nucleinic acid, a sodium salt, it will positively increase the leucocytes. That can easily be demonstrated by a certain dosage. A thirty-five-pound dog can be given as high as half a gram a day, and unless we keep it up and maintain a threefold increase of the leucocytes, at the end of ten days you have the leucocytes in the peripheral circulation. But if you go over that dose, you have a leucopenia.

As to whether the leucocytes are of any value or not, laboratory experience in my mind does not show that they are any more active than the other, but I believe they are an advantage.

If I may answer the question of Dr. Ries I will say that if you believe Allen, of England, he has never lost a case of puerperal sepsis or septicæmia because he has always given them bacterins, and they recover on the thirteenth day.

Prophylactically the typhoid bacterin has been proven of value and that has been clearly established. It will at least modify the disease, if not prevent it. It does not prevent it in animals, but that is no criterion, because we can easily protect the laboratory animals from that particular strain.

I do not agree with the doctor that bacterins are not of any value. They are of value.

DR. RIES: I did not speak generally.

DR. BIEHN: The phylacogens are bacterial toxins and will be called such before long. I used phylacogen in the case of the wife of a member of this society. She had rheumatism. He called me up and said he had used everything without beneficial effect. I told him I had used phylacogen with apparent success in two cases, but with failure in two others. He injected only one-half of the dose which is advised, and called me up about three o'clock in the morning because his wife had developed an exceedingly violent toxic nephritis. He had an awful time with her, but she pulled through, although she still has albumin and casts, and it is some three months now. She was given a second dose and it did not affect the rheumatism in any way, yet I have had apparently successful results in two cases of rheumatism.

BOOK REVIEWS

DER INFANTILISMUS, DIE ASTHENIE UND DEREN BEZIEHUNGEN ZUM NERVENSYSTEM. By Prof. Paul Mathes, M. D. Berlin: S. Karger.

In this book the author endeavors to establish the fact that infantilism and asthenia are not identical, as some authors have declared; and in the discussion of cases according to preponderance of symptoms and signs uses the names asthenic infantilism and infantile asthenia in describing them.

Anatomical conditions ("Hemmungsbilden") and the time of their appearance during the development of the individual are considered.

The author differentiates "disposition" toward disease (disease resulting from exogenous and endogenous influences) and "constitution" (organic law handed down from the parents).

In his observations from year to year of growing boys the author concludes that the lack of development in stature of different portions of the body depends upon the time at which the retardation of growth occurred, the part affected being the part which at that particular time was in active growth.

In the discussion of infantilism of the skeleton the author describes the spinal curvatures as due to the unequal development of the ventral and dorsal portions of the vertebræ, the phthisical chest as being due to lack of development of the first rib. Infantilism of the genitalia is also fully discussed, as well as the status lymphaticus and other diatheses. Asthenic enteroptosis and prolapse with the time of their most frequent occurrence are also fuliy dealt with. The second part of the book is a study of the clinical manifestations, embracing mental depression, especially before menstruation, and the physiological pathology of the sympathetic nervous system.

The book is clearly and forcefully written, showing the author's profound study of many cases and the logical conclusions drawn from them. It is a work which merits a careful translation into English.

H. A. POTTS.

ELECTRICITY IN GYNECOLOGY. By May Cushman Rice. The Practical Uses of Electricity in Diseases of Women. Second Edition. Chicago: L. I. Laing & Co.

This book is intended to supplement a course in electrotherapy. Anatomy and physics are omitted. The handling of electrodes with the technique of their preparation and use is described in detail for the purpose of assisting those already familiar with the principles involved in treating patients by electricity. These details are concisely set forth and with a moderation in results claimed unusual in a book of this nature. This is particularly evident in the chapters on uterine fibroids and on the use of the röntgen ray in carcinoma.

GOLDEN RULES OF SURGERY. By Augustus Charles Bernays, A. M., M. D., M. C. R. S. Second edition, revised and rewritten by William Thomas Coughlin, M. D. St. Louis: C. V. Mosby Company, 1913.

This little book consists exclusively of short, positive assertions, the text being classified so as to include chapters on such subjects as sepsis and asepsis, anæsthesia, shock, the operating room, general pathological conditions, and regional surgery. The two closing chapters are on "Scientific Contributions to the Literature of Medicine and Surgery" and "Science and Surgery."

One can truthfully say that there are incorporated in this brief work many excellent suggestions on surgical diagnosis and technique which, although at times owing to their positiveness might be questioned, yet for the student and general practitioner are quite in place, and will impress the reader with their importance. The chapter on "Anæsthesia," although short, is very good.

In the chapter on "Scientific Contributions to Literature" the author displays a certain amount of egotism, which not only detracts from the beauty of the sentiment with which he is endeavoring to impress the reader but rather embarrasses one's æsthetic sense.

The skilled surgeon as well as the general practitioner will find this little work not only a profitable but a pleasant evening's entertainment.

J. A. WOLFER.

HEALTH THROUGH A RATIONAL DIET. By Arnold Sorand, M. D., Carlsbad. Philadelphia: F. A. Davis.

In this book we have a most interesting and instructive lesson as regards the rational use of foods. Food is considered from every standpoint: first, as to its content of albumins, carbohydrates, and fats, and then as to the value of its salts with particular reference to phosphorus, iron, and potash. It is considered as to its energy-producing qualities and the tissue rebuilding.

Food values necessary in the different climates, in the different occupations, and in the different ages are all carefully considered. The relative merits of vegetarianism as opposed to a rational diet are carefully analyzed and the advantages of each clearly told. Food values in relation to sexual

power and reproduction are also considered to some extent.

The translation is beautifully made, and the book is read with an ease quite unusual in most books of this type. WM. R. CUBBINS.

EPIDEMIC CEREBROSPINAL MENINGITIS. By Abraham Sophian, M.D. St. Louis: C. V. Mosby Company, 1913.

Monographs of this type are welcomed chiefly by the ever-increasing class of medical men who desire to have at hand more detailed and fresher information of the advances in a definite field than can be obtained in the various systems of medicine. Cerebrospinal meningitis has in late years been the subject of much productive investigation which has yielded such valuable results both in diagnosis and in therapy that a collective report such as that embodied in this volume should meet with a warm welcome.

The author is exceptionally qualified for the presentation of this material, his association with the Research Laboratory of New York, where he carried out his own investigations on the bacteriology and serum reactions of the meningococcus, enabling him to speak with intimate knowledge of this aspect of the subject; while his experience in the Texas epidemic of 1912 yielded him a large series of observations of the epidemiological character and of the therapeusis of this disease.

The chapter on etiology, after a rather unnecessarily detailed historical preface, takes up the epidemiological aspects of cerebrospinal meningitis. The author, by an analysis of the history of the chief epidemics, is led to the conclusion that the most common predisposing agent is a period of sudden climatic changes leading to widespread occurrence in the community of a nasopharyngeal catarrh. Of 85 epidemics in the U. S. up to 1873, 37 raged in winter, 18 in winter and spring, and 23 in spring. The introduction of a virulent strain of meningococci, possibly carried over by a healthy person from a previous epidemic or carried from a recent epidemic in another country then may lead to the development of an epidemic.

The virulent strain of meningococcus may be merely implanted on a pre-existing nasopharyngitis or be the chief cause of the nasopharyngitis. The portal of entry is at all events most probably always the nasopharynx. In only certain cases, however, does further extension occur. Dopter states that it is really an epidemic of rhino-pharyngitis with which one deals — an epidemic complicated at times by meningitis which develops in a number of persons who are susceptible of cerebral invasion.

That the proportion of these susceptible cases is comparatively small is shown interestingly by the number of carriers found by bacteriological examination of the nose and throat of those who have come in contact with meningitis cases. Statistics of carrier studies in various epidemics have shown as high as 70 per cent of contacts to be meningococcus carriers.

The presence of these carriers is considered by the author to be the chief factor in the dissemination of the disease. The closing of schools and the forbidding of large gatherings are logical preventive measures. In Houston, in 1912, the visit of a well-known evangelist was followed at once by a large increase in the incidence of meningitis cases. The author points out the importance of systematic examination of the nasopharynx of all members of a family in which a case has occurred and the maintenance of quarantine until negative cultures are obtained. The methods of procedure are given at some length.

In this connection also valuable help is afforded by the discussion under the bacteriology of the meningococcus, of the other members of the gram-negative group of cocci since all of these may be encountered in throat cultures. Their differentiation by cultural characteristics is usually satisfactory. Of the various specific serum reactions Sophian considers the complement-fixation test with McNeil's specific meningococcic antigen to be at present the most reliable.

Under the symptomatology of the disease the author divides the acute form into an accumulative and an active stage. In many cases during an epidemic there is not the classical acute onset but an appreciable period of more or less definite prodromal symptoms. These, Sophian considers, correspond to the invasion of the general circulation by the meningococci from the nasopharynx with simultaneous beginning irritation of the meninges and accumulation of cerebrospinal fluid. A history of exposure, fever, headache, repeated explosive vomiting, herpespetechia, photophobia, dilated, sluggishly responding pupils, tenderness at angle of jaws, bulging fontanel, irregular pulse or respiration; a significant grouping of these may occur before the outbreak of more evident symptoms of meningitis. In no disease is early diagnosis of more value to the patient.

Flexner, in a recent article on the causation of certain accidents in the subdural injection of antimeningitis serum, speaks of Sophian as having had the largest personal experience in this country with the administration of the serum. We are prepared therefore to find as we do an unusually full and instructive discussion of this feature of the therapy. The use of simultaneous blood pressure determination in controlling the amount of serum injected is strongly and convincingly advocated as well as the use of the gravity method of injection in all except such cases as have a very thick plastic exudate. Prophylactic and therapeutic use of antimeningococcus vaccine is a new field for which the author claims attention.

The most serious defect in the book is the lack of a bibliography. The insertion of detailed quotations from the work of many men presupposes a type of

reader to whom some means of ready access to the original articles is due.

The lack of any attempt to describe the pathology of the disease is also unfortunate since it means that the book must always be supplemented and cannot serve as a complete reference to its subject.

M. C. PINCOFFS.

VICIOUS CIRCLES IN DISEASE. By Jamieson B. Hurry, M. A., M. D. (Cantab.) Second edition. London: J. & O. Churchill, 1913.

The author explains in his introduction that by the term "vicious circle" is meant a morbid process in which two or more disorders are so correlated that they reciprocally aggravate and perpetuate each other. He regards these circles as playing an important rôle in pathology, though his material, as here formulated, is entirely clinical. His effort is to discuss intelligently the pernicious influence on the progress of disease which these circles possess, as well as the large part the *ars medendi* plays in "breaking the circle." Vicious circles are first classified into what may be termed their natural orders, several examples from each order being presented.

The various systems of the body are then taken *seriatim*, so that attention may be drawn to diseases which are liable to this complication. The principal etiological factors are discussed in a special chapter. Emphasis is next laid on the necessity of breaking the circle before recovery can take place, instances being given to show how this is accomplished, both by nature and by art.

The author's aim is to give a clearer insight into problems of pathology and of rational therapeutics. Most of his material has previously appeared in the columns of the *British Medical Journal*, *The Lancet*, *The Practitioner*, and *The Medical Press*. In general, the volume presents a definite practical value, and reveals a marked philosophical tendency and a mid-Victorian condition on the part of the author.

THE MODERN HOSPITAL — ITS INSPIRATION; ITS ARCHITECTURE; ITS EQUIPMENT; ITS OPERATION. By John A. Hornsby, M.D., and Richard E. Schmidt. Philadelphia: W. B. Saunders & Co., 1913.

This very comprehensive volume is somewhat of a pioneer, as there is a dearth of literature upon the subject of modern hospital financing, building and management. The collaboration of physician and architect in the preparation of this book has given us a valuable guide to the whole hospital question whether it is designed to construct and operate a municipal or private hospital. In the introduction the authors discuss the general hospital, its division into three classes, viz., charitable, part for pay patients and part for charitable ones, and one accepting pay patients only.

Considerable space is given to financing. Plans, arrangement, details of structure, permanent installation, etc., are fully considered.

The equipment of the complete hospital as regards diet kitchens, supplies, operating rooms instruments, laundry, etc., is fully considered, and everything regarding the building and management of the hospital is advocated which makes for efficiency, economy, and care of patients. H. A. POTTS.

REPRODUCTION IN THE HUMAN FEMALE. By James Young, M. D., F. R. C. S. (Ed.) Edinburgh and London: William Green & Sons.

The investigations upon which this work is based were carried out in the Royal Infirmary and in the laboratory of the Royal College of Physicians, Edinburgh. They comprise an account of the structure of the uterine mucous membrane and the functional changes which it undergoes during menstruation and pregnancy.

The structure of the uterine mucous membrane is first described in accordance with the most recent knowledge, particular emphasis being given to the conformation of the interstitial connective tissue. The author takes an advanced position relative to the structure of the tissue network of the stroma. Many early observers regarded this method as a structural entity completely independent of the stroma, but Dr. Young attempts to show that the cell cytoplasm has been drawn on to furnish the material of which the network is formed. Again in discussing the nature of the blood supply to the corpus mucosa, he goes further than to say that blood is carried through special endothelial capillaries, affirming that the so-called blood-vessels of the endometrium are in reality simply undifferentiated tracks through the stroma. "In none of the specimens is there any evidence of muscle, fibrous, or elastic tissue in any part of the uterine stroma except that adjacent to the muscular coat. Where the vessels require extra support they derive it through a condensation of the neighboring tissue cells." "The blood-vessels, in other words, have no specialized wall, but consist merely of tracts or channels through the soft stroma protoplasm, by means of which a rich supply of blood is carried to every part of the mucous membrane." He further agrees with Leopold and Webster that the intercellular spaces in the stroma are filled with lymph.

Following rather closely in the footsteps of Hitschmann and Adler, the chapter devoted to menstruation describes the four steps of premenstrual swelling, bleeding, repair and quiescence. The subject is carried beyond a mere statement of observations, however, into an elaborate argument as to the significance of the changes now well recognized. As a result of this certain definite and advanced theories are maintained. Thus, in defining the true nature of the mucosa in terms of the functional changes which it exhibits, Young designates it not merely as a spread-out lymph surface, such as Leopold named it, but as a "potential blood sponge." Again, with reference to the causation of blood escape in the normal menstruating uterus, an interesting hint is thrown out as to the etiological

factor in certain forms of menorrhagia. Dr. Young believes that the cause of œdema and hæmorrhage in the normal mucosa is due to factors identical in nature with, and differing only in the degree of activity from, those operating in certain abnormal conditions, in which case additional support is contributed to the idea that many cases of menorrhagia and metrorrhagia are directly secondary to an abnormal activity of the ovarian influence.

Chapters three and four explain the embedding of the ovum comprising phenomena now well understood. The intra- and extrachorionic activities of the fœtal epithelium and the mode of opening up of maternal vessels and of the formation of blood sinuses is described in detail. Some consideration is given also to the question of placental fragments and to chorio-epithelioma. The author maintains that an escape of blood by merely mechanical means from a broken-down vessel wall does not suffice to explain the manner in which the fœtal elements derive their nourishment. Hence he speaks of a "chorionic influence" which "in some way or other determines the occurrence of protoplasmic changes in the cells, which lead to their imbibing fluid which can often be seen to have accumulated in the cell body under a hydrostatic tension clearly higher than the pressure of the intravascular blood and of the fluid in the surrounding tissue spaces.

The Tescher-Bryce ovum and those of Peters, of Jung, and of Leopold are briefly described, followed by a detailed report of the author's own specimen. For this he estimates an age of 13½ to 14½ days, thus placing it between the Tescher-Bryce and the Peters ova in measurements.

The nature of the chorionic influence and the mode of its spread is discussed briefly without, however, the development of any very definite theory as to its chemistry. The changes taking place in the endometrium in the formation of the decidua are entered upon in detail, as is a discussion as to the functions of this membrane. Concerning this function the author says:

"The function of the decidual membrane is to prevent an irregular and increasing opening-up and expansion of the maternal vessels and a tearing-up of the mucosa by fluid and blood after the tissue changes necessary for an adequate supply of the maternal blood for the engrafted ovum have been accomplished. It is by virtue of this action that the chorionic invasion of the tissue is limited and that the increase in the implantation chamber is prevented by an erosion or displacement of tissue."

In some respects the most important feature of this volume is the collection of plates and microphotographic reproductions. These are excellent in character, abundant in number and unusually well reproduced. They not only serve to render the text more understandable—of definite value in a work of such specialization as this—but, to those familiar with the more recent studies in placentation, such reproductions are of greater value than any teacher's theories.

In stating that for the first time it is shown that the structural conformation of the uterine mucosa is intimately bound up with menstruation and with the processes associated with fœtal nutrition the author apparently claims overmuch, considering alone Webster's very fundamental work on placentation in 1901 and the exposition of Hitschmann and Adler's research in 1907. Yet, coming at this time, the present volume shows genuine research and is of a definite value as an addition to the literature of the subject. While the author goes beyond mere demonstration of findings and attempts to establish advanced theories, it must be said that his theories reveal scientific imagination. In most respects they are logical in their deduction and attractive in their presentation.

CAREY CULBERTSON.

MUSCLE SPASM AND DEGENERATION IN INTRATHORACIC INFLAMMATIONS AND LIGHT TOUCH PALPATION. By Francis Marion Pottenger, A. M., M. D., LL. D., Medical Director of the Pottenger Sanatorium for Diseases of the Lungs and Throat, Monrovia, Cal. St. Louis: C. V. Mosby Co., 1912.

This volume is designed to promulgate, according to the author's methods and observations, our knowledge regarding the diagnosis of diseases of the thoracic organs. He lays great stress upon inspection and palpation, especially light touch palpation, which methods have heretofore not been recognized as being of especial value as diagnostic aids in intrathoracic diseases. According to the observations and studies by the author those diseases when acute produce a spasm of overlying muscles which degenerate when the process becomes chronic. Even in a degenerated muscle, spasm is produced by an acute exacerbation of a chronic process, as frequently is the case in pulmonary tuberculosis. Many of the facts presented are not new and the only claim the author has to originality is that he has worked them out as an independent observer and has applied them to clinical cases to his own satisfaction as to their diagnostic aids.

The author discusses the cause of this degeneration, further outlining what can be determined by noting the condition of the neck and chest muscles in acute and chronic inflammations of the lungs and pleura as well as the relation this muscle change bears to the bony anomalies of the upper thorax.

The light touch palpation and the application of it in the delineation of the deep-seated organs is distinctly the author's, and if it can be developed by the average diagnostician to the degree which the author has attained it will mark a distinct advance in clinical diagnosis.

H. A. POTTS.